

**THE UNIVERSITY OF HULL**

**The Effects of the Glycaemic Index of Carbohydrate Meals on  
Metabolism, Recovery and Endurance Performance.**

**being a Thesis submitted for the degree of Doctor of  
Philosophy in the University of Hull**

**by**

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**PUBLISHED RESEARCH AND RESEARCH SUBMITTED FOR  
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**L.J.S. Moore**, A.W. Midgley, L.R. McNaughton, and J. Siegler. The effect of high and low GI recovery meals during intermittent, high intensity exercise on subsequent endurance performance. (Under review)

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## LIST OF ABBREVIATIONS AND ACRONYMS

|      |                               |
|------|-------------------------------|
| ADP  | adenosine diphosphate         |
| ATP  | adenosine triphosphate        |
| CHO  | carbohydrate                  |
| cm   | centimetres                   |
| d    | day                           |
| GI   | glycaemic index               |
| h    | hour                          |
| HGI  | high glycaemic index          |
| kg   | kilograms                     |
| km   | kilometres                    |
| L    | litres                        |
| LGI  | low glycaemic index           |
| m    | metres                        |
| min  | minute                        |
| mL   | millilitres                   |
| mm   | millimetres                   |
| mM   | millimolar                    |
| mmol | millimole                     |
| n    | number of subjects in sample  |
| PRO  | protein                       |
| RER  | respiratory exchange ratio    |
| RPE  | ratings of perceived exertion |
| s    | seconds                       |
| SD   | standard deviation            |
| TT   | time trial                    |

$\mu\text{L}$

microlitres

W

watt

## LIST OF SYMBOLS

|                                  |                       |
|----------------------------------|-----------------------|
| $\text{CO}_2$                    | carbon dioxide        |
| $\text{HR}_{\text{max}}$         | maximal heart rate    |
| $\text{HR}_{\text{peak}}$        | peak heart rate       |
| $\text{O}_2$                     | oxygen                |
| $\dot{\text{VCO}}_2$             | carbon dioxide output |
| $\dot{\text{VO}}_2$              | oxygen uptake         |
| $\dot{\text{VO}}_{2\text{max}}$  | maximal oxygen uptake |
| $\dot{\text{VO}}_{2\text{peak}}$ | peak oxygen uptake    |

## ABSTRACT

The aim of this thesis was to investigate the effect that consuming pre- and post exercise meals, varying in GI, would have on physiological responses and subsequent endurance performance.

Study 1 investigated the effects of consuming either a low or high GI meal 45 min prior to exercise on cycling TT performance. It was observed that consuming a low GI meal 45 minutes prior to performing a 40k TT, resulted in a significantly improved performance ( $p = 0.009$ ) compared to the ingestion of an isocaloric high GI pre- exercise meal ( $93 \pm 8$  min vs.  $96 \pm 7$  min for low and high GI, respectively). The results suggested that the ingestion of the low GI food led to an increase in the availability of CHO and greater CHO oxidation throughout the exercise period and subsequently spared limited muscle and liver glycogen stores.

Study 2 investigated the effects of low and high GI 24 h diets, following glycogen depleting exercise, on TT performance the following day. No difference was observed in TT performance following low ( $90.7 \pm 11.1$  min) and high ( $93.5 \pm 9.29$  min) GI, 24 h recovery diets ( $p = 0.35$ ). This study concluded that provided the amount of CHO consumed during the recovery period is sufficient enough to replenish depleted muscle glycogen stores, the GI of the recovery diet offers no further benefit to performance.

Study 3 investigated the effects of high and low GI recovery meals on TT performance following a short term recovery period from a glycogen depleting exercise. No significant difference was observed in TT performance between the low ( $90.7 \pm 11.1$  min) and high GI ( $93.5 \pm 9.3$  min) trials ( $t = 1.1$ ;  $p = 0.35$ ). This study concluded that if the following exercise bout is of short duration, and not long enough to challenge glycogen stores, provided the amount of CHO is sufficient during the recovery period, the GI of short- term recovery diets has no influence on subsequent exercise performance.

In conclusion, the findings presented in this thesis should contribute to and support previous research within this area and help to contribute to the body of knowledge through greater ecological validity.

**Key words:** glycaemic index, carbohydrate oxidation, endurance performance, physiological responses, muscle glycogen.

# Chapter 1

## Introduction and Review of Literature

### 1.0 Introduction:

The food that we consume supplies both the structural elements of the body and the means of sustaining the body's energy requiring processes by providing the fuels and building materials for life. Energy is required for all biosynthetic pathways and for the preservation of the internal environment of the body (Maughan and Burke, 2004). Once the body's basal needs have been met, additional energy is required to fuel occupational, recreational, or sporting muscular activity. All this muscular activity places an increased energy demand on the muscles, which if not met, cannot be performed. As the exercise intensity increases or the duration is prolonged, there may be difficulty in supplying energy at the required rate, and fatigue develops (Hargreaves, 2000).

During increasing levels of energy expenditure, the demand for energy is increased as is the demand for associated nutrients for increased metabolism. Although, in essence, the dietary needs of the athlete are fairly similar to those of the average individual, differences may arise due to increased energy needs, the timing of meals to ensure an adequate intake around a busy schedule, and the importance of maintaining a high intake of carbohydrate (CHO) during training and after competition (Barasi, 2003).

The fuel sources that power exercise are generally dependent on the intensity and duration of effort as well as the nutritional and training status of the athlete. The sources for energy transfer exist along a continuum. The intramuscular high- energy phosphates adenosine triphosphate (ATP) and phosphocreatine (PCr) systems provide approximately one half of the energy required for intense exercise lasting 2 minutes, with aerobic reactions providing the remainder. Intense exercise of intermediate duration performed for 5 to 10 minutes requires a greater dependency on aerobic



energy transfer. Performances of a longer duration require a steady energy supply derived aerobically without reliance on lactate formation.

The immediate energy source for actin- myosin cross- bridge cycling during exercise is ATP. It is therefore essential that a constant supply of ATP is maintained. Because the intramuscular concentration of ATP is small (5- 6mmol. kg.ww<sup>-1</sup>) (Hargreaves, 2000), ATP must be derived from other metabolic pathways in order to maintain the rate of ATP resynthesis necessary for ongoing contractile activity. The energy released in macronutrient break down is crucially used to phosphorylate ADP to re- form ATP. Macronutrient catabolism favours the generation of ATP, however, the specific pathways of degradation differ depending on the nutrients metabolised. When movement begins at either fast or slow speed, the intramuscular high- energy phosphates provide immediate anaerobic energy for muscular contraction. After a few seconds, the glycolytic pathway (intramuscular glycogen breakdown in glycolysis) generates an increasingly greater proportion of energy for ATP re- synthesis. Intense exercise continued beyond 30 sec places a progressively greater demand on the relatively slower aerobic energy metabolism of the stored macronutrients. This anaerobic glycolysis releases only a bout 10% of the energy within the original glucose molecule, therefore additional metabolic pathways are used to extract the remaining energy. This occurs when pyruvate irreversibly converts to acetyl- CoA, which then enters the second stage of CHO breakdown known as the Krebs cycle. The process of converting potential energy in fatty acid to ATP is called beta oxidation. Beta oxidation provides the same two- carbon acetyl unit as acetyl generated from glucose breakdown. Beta oxidation continues until the entire fatty acid molecule degrades to acetyl CoA and enters the Krebs cycle directly. Fatty acid breakdown is directly related to oxygen uptake, which is sometimes a limiting factor in the use of fatty acids as a preferred fuel choice (Jeukendrup and Gleeson, 2004).

Carbohydrates are a group of substrates, which are found in both plants and animals and are composed of carbon, hydrogen and oxygen (Barasi, 2003). Carbohydrates used to be regarded as of little importance in the diet, supplying only energy. However, since the early 1930's, the importance

of CHO in an individual's diet has been recognised, and these have been therefore been promoted as a major source of energy and nutrients (Burke, 2003). The importance of CHO for exercise performance has been recognised since the early classic respiratory exchange studies of Christensen and Hansen (1937) and Bergstrom *et al.* (1967), who measured muscle glycogen during various dietary and exercise interventions. Since then, considerable attention has been placed on nutritional strategies to maximize endogenous CHO (liver and muscle glycogen), so minimizing the potential ergolytic effects of CHO depletion (Coyle, *et al.*, 1986).

Fatty acids are essential components of cell walls and intracellular membranes, therefore, all living cells contain some form of fat in their structure. Fat is essential to body composition and, as a result, has several diverse functions within the body including structure, storage and metabolism (Barasi, 2003). The provision of energy, however, is probably the most recognized function of fats. Fat is an energy- dense fuel, providing more energy per gram than that provided by both CHO and protein per gram (Frayn, 1996). This is important as it enables individuals to store a large amount of reserve energy in a relatively small volume. The body utilises stored fat as its major energy supply, releasing non- esterified fatty acids (NEFA) into the circulation under the influence of noradrenaline, which is released by the sympathetic nervous system (Frayn, 2003). At most levels of activity, fat provides a significant proportion of the energy used. However, once the intensity of the exercise increases (e.g. running the 100m sprint) the energy predominantly comes from CHO, since fat cannot be metabolized at an appropriate pace (Hargreaves, 2000). The lower the intensity of the activity, the greater the production of energy comes from fat (Figure 1.1). However, some glucose continues to be used for metabolism as the brain, nervous system, and red blood cells, require it.

Fat sources (plasma free fatty acids derived from adipose tissue and intramuscular triglycerides) are relatively plentiful, however CHO sources (plasma glucose derived from the liver or dietary CHO intake, and muscle glycogen stores) are limited (Barasi, 2003, Burke, 2001). Whatever the intensity of the exercise, both fat and CHO is generally used (Spriet *et al.*, 1987).

Eventually, the supply of CHO is depleted and exercise will have to stop. As a result, sports nutrition guidelines have focused on strategies to enhance body CHO availability. Such guidelines include the intake of CHO before and during exercise to provide fuel for that session, as well as the consumption of CHO after the session and over the day in general to enhance refuelling and recovery.

## **Literature Review:**

### **1.1 Macronutrient storage:**

Carbohydrates and fats provide the majority of the dietary energy, and glucose and fatty acids are the main form in which they are distributed throughout the body (van Loon, 2004). These metabolic fuels are used to resynthesise ATP through various metabolic and energy pathways. However, as the central nervous system and some tissues and organs cannot oxidise fatty acids, glucose must therefore be supplied so that these energy needs can be met. Protein provides only a minimal amount of dietary energy, body weight maintenance and energy provision is determined predominantly by the intake and utilisation of CHO and fat (Flatt, 1995).

Endogenous glycogen stores are relatively small and generally range between 460 and 520g, which correspond to a total energy storage of 1785- 2000kcal (van Loon, 2004). Carbohydrate cannot be synthesised from the more abundant store of fat within the body. The body must, therefore, maintain a store of carbohydrate. Carbohydrates are hydrophilic. Therefore, when CHO is stored in cells it is stored in a hydrated form, in association with water and is always associated with approximately three times its own weight of water (3g. water.kg<sup>-1</sup>). This reduces the energy density of the glycogen stores to approximately 1kcal.g<sup>-1</sup>) (Flatt, 1995), which clearly limits the amount of energy that can be carried in the form of glycogen.

Fat is mainly stored as triacylglycerol (TG) in subcutaneous and deep visceral adipose tissue. Smaller quantities of TG are present as lipid droplets inside muscle fibers (IMTG). In

contrast to the endogenous CHO deposits, endogenous fat stores are quite large and range between 9 and 15kg, corresponding to a total energy storage of 80,000- 140,000kcal, which is sometimes 60 times higher than that stored under the form of glycogen (1,700- 2,000kcal) (Horowitz and Klein, 2000; van Loon, 2004). IMTG contents have been shown to be quite variable and are approximately ~0.2 kg, which corresponds to a total energy storage of 1850kcal (van Loon, 2004). In contrast to the aqueous environment of glycogen, fat is stored as a lipid droplet from which water is excluded. Mainly because of this lack of water, fat stores contain considerably more energy per unit weight ( $\sim 8\text{kcal.g}^{-1}$ ) of store than do CHO stores (Flatt, 1995). This allows us to accumulate and store much more energy as fat, due to its high energy density than that, which is possible for CHO.

Amino acids can be oxidised to provide energy, or converted to glucose and fatty acids, which can be oxidised. However, an average PRO store is around 12.5kg, which, when completely oxidised, amino acids only liberate around  $24\text{kJ.g}^{-1}$ , which is not enough, as an independent fuel store, to support continuous metabolic oxidation. These data presented above clearly stress the importance of these fuels in terms of storage and their relevance to exercise performance, which will be described in more detail in the following sections.

## **1.2 Fuel availability and utilization:**

Energy is required by all muscle cells to perform work and can only be used in the form of what is known as ATP. All the energy requiring processes within the muscles are powered by the energy released when this ATP is converted to ADP with the release of an inorganic phosphate group ( $\text{P}_i$ ) (Jeukendrup and Gleeson, 2004). As previously mentioned, there is only a small amount of ATP within the muscle cells, amounting to approximately  $\sim 5\text{mmol.l}^{-1}$  (3.4g), which in the average healthy male with ~20kg skeletal muscle, is about 70g of ATP in total (Hargreaves, 2000). Most activities only utilise a minimal amount of the total muscle mass and therefore, the actual amount of ATP available is much less. Since the content is so small and must be maintained, the ATP that is

within the muscle cannot be considered as a usable energy store (Maughan and Burke, 2004). The muscle does however, have a second source of readily available energy in the form of CP, which is present within the muscle at a concentration which is 3-4 times the amount of ATP (14- 24mmol. ATP.kg.ww<sup>-1</sup> vs. 5- 6mmol. ATP.kg.ww<sup>-1</sup> for CP and ATP, respectively (Jeukendrup and Gleeson, 2004). Although the muscle concentration of ATP is closely monitored and are typically only reduced by 30- 50% (Soderlund and Hultman, 1991; Spreit *et al.*, 1989) that of CP can be completely depleted following a few seconds of maximal exercise (Soderlund and Hultman, 1991). The resynthesis of ATP from ADP by transfer of a phosphate group from CP is extremely rapid, and can supply energy at very high rates (2.25mmol. ATP.kg.ww<sup>-1</sup>.s) and a subsequent high power output (2.3mmol. ATP.kg.ww<sup>-1</sup>.s) (Jeukendrup and Gleeson, 2004), however, this high capacity is limited and this level cannot be sustained for long. Therefore, for more prolonged exercise, an alternative fuel must be utilised.

Glycogen, which consists of very large branched polymers of glucose, is stored within the muscle cells, and glycogenolysis (catabolism of glycogen) can supply relatively large amounts of energy to the muscle (Jeukendrup and Gleeson, 2004). Glycogen exists in two forms in human muscle (Lomako, Lomako and Whelan, 1990). Macroglycogen is the larger, acid soluble form and Proglycogen is the smaller, acid insoluble form (Adamo, Tarnopolsky, Graham, 1998). Studies have suggested that the rates of accumulation and utilization of both forms of glycogen are different depending on the mixed muscle glycogen concentration and other metabolic factors (Adamo *et al.*, 1998; Graham *et al.*, 2001). The amount of available energy from the glycogen store is large and highly variable. If glycogenolysis proceeds only as far as lactate, then the muscles can produce 2 molecules of ATP for each mol of glucosyl units consumed (Frayn, 1996). This series of reactions, although rapid, produces ATP at a rate (1.10mmol.ATP.kg.ww<sup>-1</sup>.s) that is less than that supplied from CP. If glycogenolysis is allowed to go on to completion with the oxidation of pyruvate through the tricarboxylic acid cycle (TCA cycle) to carbon dioxide and water, then the amount of ATP produced is much greater, being 39 molecules per mol of glycosyl units (Hargreaves, 2000).

Although the amount of energy ( $48\text{--}75\text{mmol.ATP.kg.w}^{-1}.\text{s}$ ) that can be liberated from glycogen stores is increased, the rate at which it is available is much less (Jeukendrup and Gleeson, 2004).

An alternative source of CHO fuel available to the muscles is blood glucose, which is derived from the liver. The total amount of glycogen stored in the liver varies greatly and a value of 80g has been reported (Maughan and Burke, 2004). The liver glycogen content can fall by more than 50% following a 12h fast, suggesting that the liver is important in the maintenance of the blood glucose concentration. To do this, the liver relies on glucose 6- phosphate, which releases free glucose, which is then able to leave the cells. Within muscle, this enzyme is absent and therefore, the sugar phosphates that are formed when glycogen is broken down are trapped within the cells (Salway, 1995). If glucose, as opposed to glycogen is the starting point, then one ATP molecule is required in order to convert the glucose to glucose 6- phosphate, so the net gain in energy to the cell is 3 molecules of ATP for each glucose molecule broken down, compared to the 4 molecules when glycogen is the starting point (Frayn, 1996). This, together with the reduction on the rate at which glucose can enter the muscle cells, makes glycogen a more suitable fuel when the energy demands placed on the body are high.

By manipulating the diet and exercise patterns of an individual, the glycogen content of both the muscle and the liver can vary widely (Zderic *et al.*, 2004). In addition to the liver's store of glycogen, it can also release glucose that has been produced by the process of gluconeogenesis. In gluconeogenesis, glucose is synthesized from other substrates, which can include lactate, amino acids, and glycerol derived from triglycerides (Hargreaves, 2000). Glucogenesis can account for more than half of the glucose, which is released from the liver in some situations, and may be particularly important in prolonged exercise when glycogen stores are low during exercise.

In addition to the substrates mentioned above, muscle also has the ability to obtain energy from the oxidation of fat in the form of free fatty acids (FFA). Free fatty acids are derived from triglyceride, as each triglyceride molecule can be broken down to release three molecules of fatty acids and one of glycerol (Frayn, 1996). The body stores large amounts of triglycerides within

adipose tissue and within the muscle itself but to a much smaller extent. However, unlike the body's CHO stores, the body's fat stores are not susceptible to large changes in the short term (Maughan and Burke, 2004). The rate at which energy can be produced from fat oxidation is, however, much less than can be achieved through CHO oxidation. The amount of energy that is released per unit of oxygen consumed is much less for fat ( $19.7\text{kJ} \cdot \text{L O}_2^{-1}$ ) than for CHO ( $21.4\text{kJ} \cdot \text{L O}_2^{-1}$ ). However, fat is beneficial as a fuel as it is an extremely efficient storage form and releases approximately  $37\text{kJ} \cdot \text{g}^{-1}$  compared to  $16\text{kJ} \cdot \text{g}^{-1}$  for CHO (Frayn, 1996). All of these above fuels are utilised by the muscle in order to produce energy during exercise. The proportions in which they contribute to the total energy requirement will depend on several different factors including the intensity and duration of the exercise, nutritional status of the individual, and the physiological and biochemical characteristics of the individual (Hargreaves, 2000).

#### *Anaerobic and aerobic metabolism*

Anaerobic exercise is characterised by sprinting or weight- lifting, it is of short- duration, but may also involve great strength and be dominated by the activity of the fast twitch (Type II) muscle fibres (Pette and Spamer, 1986). Muscle contraction is maintained by continued small contractions of individual muscle fibres, and there must be a continued stimulation of the muscle by the somatic nerves and sustained ATP production within the muscle to maintain a contraction. The main feature of anaerobic exercise is the rapid production of energy over a short period of time. Energy is generated too rapidly for the diffusion of substrates including oxygen, from the blood, into the muscle and therefore, energy production is achieved by the utilization of the muscle's own energy stores, phosphocreatine and glycogen (Frayn, 1996).

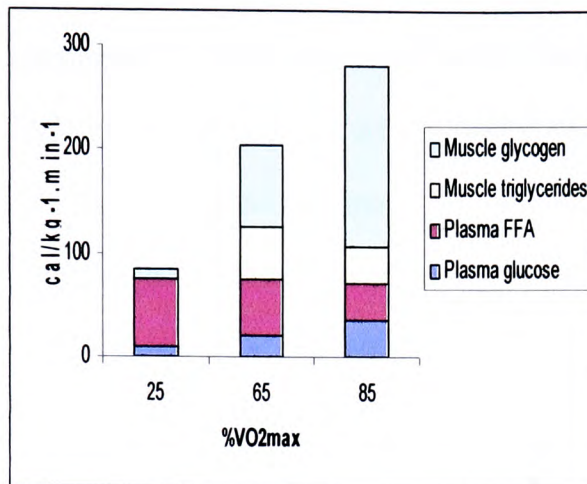
Aerobic exercise is characterised by prolonged exercise performed at a lower intensity than could be achieved anaerobically. The muscle fibres involved are predominantly the oxidative, type I fibres (Pette and Spamer, 1986). For this type of exercise, the duration is such that it cannot be supported entirely from fuels stored within the muscles, so the fuel stores in the rest of the body (fat in adipose tissue and liver glycogen) must be utilised. These substrates are brought to the muscle in

the blood, following necessary adjustments, which are made to the circulatory system (Frayn, 1996). This type of exercise is called aerobic because, to maximize efficiency, these substrates (fatty acids and glucose) are completely oxidised. In order for the muscles to produce external work at a high rate over a long duration, they must be supplied with substrates, including  $O_2$ , and the waste products of metabolism, such as carbon dioxide ( $CO_2$ ), must be removed, at a sufficiently high rate (Jeukendrup and Gleeson, 2004). This thesis is mainly concerned with aerobic exercise and the main fuel sources utilised by the muscles in order to produce energy during this aerobic type of exercise.

During prolonged exercise at a constant intensity, the muscle predominantly relies on oxidative metabolism to produce energy. The exception to this is the first few minutes of exercise when anaerobic metabolism will contribute to energy production until typically a steady state of oxygen consumption is achieved (Hargreaves, 2000). Oxidative metabolism provides energy for the resynthesis of ATP and CP and to remove lactate during the recovery periods. The major fuels contributing to oxidative metabolism during prolonged exercise are fat and CHO (Brooks, 1991). Protein oxidation only accounts for approximately 5% of the total energy requirement in exercise lasting up to 2-3h (Frayn, 1996). The relative contributions of fat and CHO are largely determined by both the intensity and the duration of the exercise being performed (Rankin, 1997). During low intensity exercise at approximately 50% of an individual's  $\dot{V}O_{2\max}$ , fat is the predominant fuel and accounts for more than half of the total energy produced, with the oxidation of blood glucose and muscle glycogen contributing equally to the remainder (Coyle, 1995). At about 60- 65% of an individual's  $\dot{V}O_{2\max}$ , the contributions of fat and CHO are roughly equal, and anything above this level of exercise, CHO is the predominant fuel (Coyle, 1995). In moderate- intensity exercise corresponding to about 70- 75% of  $\dot{V}O_{2\max}$ , which can usually be sustained for 2-4h, muscle glycogen will be the major fuel (Hargreaves, 2000). At higher exercise intensities, the rate of CHO utilization will continue to increase, and once the muscle has to rely on anaerobic glycolysis to



supplement the energy produced by oxidative metabolism, the rate of glycogen utilization greatly increases (Figure 1.1)



**Figure 1.1: Contributions of fuel to energy production during exercise of increasing intensity (Romijn *et al.*, 1993).**

Typically, CHO use increases concomitantly with intensity and decreases with prolonged duration of an activity. The absolute amount of CHO and fat used by muscles, however, can be shifted, depending on exercise intensity and fuel availability, in that the greater the availability of fatty acids the greater the use of fat (Lambert *et al.*, 1997; Pitsiladis *et al.*, 1999). Similarly, when more CHO is available, more CHO will be metabolised for energy (Rankin, 1997). In contrast, the availability of fatty acids to the muscles will also reduce the rate of glucose oxidation via the glucose fatty acid cycle as there is evidence to show that increasing the availability of fatty acids leads to sparing of glycogen, which allows high- intensity exercise to be maintained for longer (Stevenson *et al.*, 2005; Wu *et al.*, 2003). However, several studies have shown that fat oxidation can only support around 60% of the maximal aerobic power output (Stisen *et al.*, 2006). In ultra-endurance athletes (e.g. 24h runners), power output drops with time to about 50% of maximal aerobic power, which is roughly the same time as the glycogen stores would be expected to be depleted. In less well- trained individuals, fat oxidation contributes to a maximum of about 60% of

muscle oxygen consumption (Romijn *et al.*, 1993) and therefore, for the maintenance of maximal aerobic power, CHO will have to be oxidised as well as fat (Figure 1.1).

As this CHO comes from the glycogen stores within the body, the time for which maximal aerobic power can be sustained is dependent on the initial amount of glycogen stored. Depletion of the glycogen stores will lead to a sudden feeling of fatigue, which has been demonstrated in early research by Bergstrom and Hultman (1967). They measured the content of glycogen in small muscle biopsies, in a group of athletes who were each studied on two or three occasions after the ingestion of different diets. These different diets (mixed; low CHO; high CHO) initially produced varying concentrations of muscle glycogen, and it was found that the time to exhaustion, when working at 75%  $\dot{V}O_{2\max}$ , correlated with the initial muscle glycogen concentration. This observation led to the development of methods aimed at boosting the muscle glycogen stores for endurance runners, such as glycogen/ CHO loading.

Given what is known about fuel availability and utilisation during moderate- to high intensity exercise, the aims of dietary intervention for endurance athletes should be to maximise CHO (glycogen) stores in the muscles and liver and to make both CHO and fat readily available in the blood for use by the working muscles. The metabolic challenge should therefore be to maintain CHO supply to the muscle but to also slow its depletion by relying optimally as far as a fuel. The aim of consuming food prior to exercise should be to supply CHO that can elevate or maintain blood glucose without massively increasing insulin secretion. This would theoretically optimize the availability of both glucose and FFA's for use by the working muscles.

### **1.3 Carbohydrate ingestion:**

Many studies have investigated the ergogenic value of consuming CHO before (Neufer *et al.*, 1987; Sherman *et al.*, 1989), during (Burke *et al.*, 1998; Jentjens *et al.*, 2003; Wright, Sherman and Dernbach, 1991), or after (Bowtell *et al.*, 2000; Burke, Collier, and Hargreaves, 1993; Ivy *et al.*, 2002) an exercise bout and there is a plethora of evidence to suggest that CHO consumption before

and/ or during prolonged exercise can significantly enhance endurance performance (e.g. Bergstrom *et al.*, 1967; Burke *et al.*, 1999; Coyle *et al.*, 1986).

Only two tissues, skeletal muscle and liver, have significant stores of glycogen in relation to the requirements of the whole body, although the majority of tissues have a small store for local use (Frayn, 1996). Approximately 40% (25kg) of the human body is comprised of skeletal muscle (in a 70kg man), and a typical concentration of glycogen in skeletal muscle is around  $15\text{g.kgww}^{-1}$ , contributing to a total muscle glycogen store of ~ 350- 500g (Frayn, 1996; van Loon, 2004). This, however, is not all available directly as glucose to enter the circulation, since muscles lack glucose-6 phosphatase only one free glucose molecule is released as a  $\alpha$  (1-6) glucosidase, it can, however, be exported to the liver as lactate, pyruvate, and/ or alanine for the formation of glucose (glucogenesis)(Salway, 1995). In contrast, the liver glycogen store is more directly available as glucose, and is used as a buffer for changing hour- to- hour energy requirements. A typical liver glycogen concentration is ~50- 80g.kgww<sup>-1</sup>, which varies during the day and as the liver weighs ~1- 1.5kg, the total liver glycogen store is ~50- 120g (Maughan and Burke, 2004).

The main priorities of pre- exercise CHO feedings are to increase the glycogen content of the liver or skeletal muscle as well as provide an absorbable source of CHO as it empties from the stomach (Wright *et al.*, 1991). Therefore, athletes are recommended to consume diets that can provide 7- 10g.kgbw<sup>-1</sup> of CHO(Burke *et al.*, 1995; 2001; Costill *et al.*, 1981), which, if not met can cause the athlete to compete with below normal muscle glycogen content, which as a result, may negatively affect their athletic performance (Wright *et al.*, 1991).

Given that CHO availability is enhanced by increasing muscle and liver glycogen stores, the latter of which being more susceptible to depletion by an overnight fast (Frayn, 1996), pre- exercise CHO intake on the morning of an event may be important for maintaining blood glucose levels via hepatic glucose output during the latter stages of prolonged exercise. This is especially important for athletes who train or compete following an overnight fast who may fatigue prematurely because of an untimely decline in blood glucose (Coyle and Coggan, 1984) due to significantly reduced liver glycogen stores following the overnight fast. Compared to results achieved after an overnight fast,

the intake of a substantial amount of CHO (~200-300g) two- four hours prior to exercise has been found to prolong cycling endurance (Wright *et al.*, 1987) and enhance performance of an exercise test undertaken following a standardised cycling task (Neufer *et al.*, 1987; Sherman *et al.*, 1989).

The aim of consuming a meal before exercise is to contribute additional CHO for oxidation and subsequently spare limited muscle and liver glycogen stores (Sherman *et al.*, 1991). The oxidation of ingested CHO has been investigated using stable and radioactive isotope techniques and several authors have shown that there appears to be an upper limit to which CHO can be absorbed and oxidized of about  $1\text{g}\cdot\text{min}^{-1}$  (Hawley, Dennis and Noakes, 1992; Jeukendrup *et al.*, 1996; 1999). When large amounts of glucose are ingested ( $>1.2\text{g}\cdot\text{min}^{-1}$ ), intestinal glucose transporters (SGLT1) may become saturated making intestinal CHO absorption a limiting factor for exogenous CHO oxidation (Jeukendrup and Jentjens, 2000; Jeukendrup, 2004). It has therefore been recommended, based on studies that show performance enhancements that CHO should be ingested at a rate of about  $60\text{g}\cdot\text{hour}^{-1}$ . Conversely, as previously mentioned, a high- CHO meal is known to stimulate CHO oxidation in the initial period of exercise, which may stimulate greater rates of glycogenolysis and a subsequent early depletion of glycogen stores. Glycogen depletion, which leads to hypoglycaemia, is suggested to be the primary cause of fatigue during prolonged bouts of exercise lasting more than 60- 90min duration (Burke *et al.*, 2004; Coyle *et al.*, 1986; Hermansen *et al.*, 1967; Kirwan *et al.*, 1988). Therefore, the utilisation of fuel between endogenous and exogenous sources of CHO after a CHO- rich meal is critical for optimal exercise performance.

Although there are recommendations about the amount and frequency of CHO ingestion before, during and after exercise, these recommendations typically do not include any comment on the specific type of CHO that should be consumed.

#### **1.4 The metabolic challenge:**

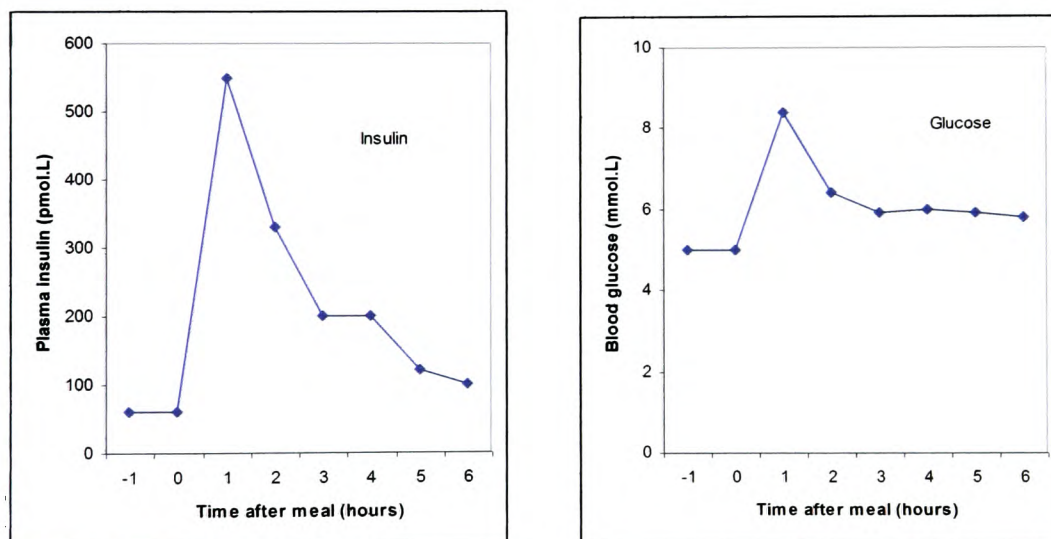
As previously mentioned, the ability to sustain prolonged aerobic exercise is determined largely by fuel availability. Although, a low muscle glycogen concentration and an insufficient rate of ATP resynthesis can all have an influence on performance, Christensen and Hansen (1939) were the first

to notice a relationship between hypoglycaemia and fatigue. These authors demonstrated that maintaining euglycemia and CHO oxidation in the latter stages of exercise can delay fatigue. This finding promoted the investigation into ways in which euglycemia can be maintained during exercise in order to optimize exercise performance. Additionally, CHO ingestion both prior to and during prolonged exercise, which prevents a hypoglycaemic (low blood glucose) response, may result in improved performance by increasing the availability of glucose to the working muscles. Increasing the availability of this substrate should lead to an increase in the blood glucose oxidation when intramuscular glycogen stores become compromised (Coyle *et al.*, 1985; Neufer *et al.*, 1987; Sherman *et al.*, 1991; Wright *et al.*, 1991). Research by Sherman *et al.* (1991) for example, demonstrated that consuming either  $1.1\text{g}\cdot\text{kgbw}^{-1}$  of liquid CHO or  $2.2\text{g}\cdot\text{kgbw}^{-1}$  of high CHO meal prior to cycling at  $70\% \dot{V}\text{O}_{2\text{max}}$ , followed by a performance trial, significantly improved time trial performance by 13.1% compared to a placebo ( $P < 0.05$ ). Additionally, Neufer *et al.* (1987) found that consuming 45g of either liquid or solid CHO, in both the fed and fasted state, 5 min prior to cycling at  $70\% \dot{V}\text{O}_{2\text{max}}$ , significantly enhanced total work produced compared to a placebo ( $175 \pm 12$ ,  $176 \pm 10$ ,  $159 \pm 11\text{N}\cdot\text{m}^{-1}$ , respectively,  $P < 0.05$ ). An additional trial was performed, in which a high- CHO meal (200g) was consumed 4 h before exercise, combined with the ingestion of a confectionary bar immediately before the exercise. This trial resulted in significantly greater work produced compared to all the other previously mentioned trials ( $194 \pm 4\text{N}\cdot\text{m}^{-1}$ ,  $P < 0.05$ ). As a result of this, and other research (Burke and Hawley, 1999; Ivy, 1999; Kirwan *et al.*, 1988), it is now widely accepted that CHO ingestion before, during, and in recovery from exercise makes an essential contribution to substrate availability and in most cases leads to enhanced exercise performance and recovery from strenuous exercise. However, despite these consistent data on CHO ingestion during prolonged exercise, the issue of pre- exercise feeding on metabolism and exercise performance still remains unclear. In fact, this practice has been demonstrated to increase (Coyle *et al.*, 1983; Sherman *et al.*, 1991; Thomas *et al.*, 1991; Wright *et al.*, 1991), decrease (Foster *et al.*, 1979), or have no effect (Devlin *et al.*, 1986) on exercise performance. Coyle *et al.* (1983) demonstrated that consuming a glucose polymer prior to cycling to fatigue at  $74 \pm 2\%$  of  $\dot{V}\text{O}_{2\text{max}}$ ,

resulted in 20- 40% higher blood glucose concentrations and significantly longer cycle times to exhaustion when compared to a placebo  $157 \pm 5$ , and  $136 \pm 6$ min, respectively,  $P < 0.01$ ). These findings were later corroborated by Wright *et al.* (1991), who provided their participants with one of four test meals prior to cycling to exhaustion at  $70\% \dot{V}O_{2\max}$ . The participants were provided with CHO either pre- exercise, during exercise, before and during exercise, or not at all (placebo). At exhaustion, glucose had declined to 4.0, 4.6, 5.0 and  $3.8 \text{ mmol} \cdot \text{l}^{-1}$  and time to exhaustion was 18%, 32% and 44% greater, respectively, than the placebo group ( $p = < 0.05$ ). The authors postulated that the improvements in performance following the ingestion of CHO before and/ or during exercise, was probably the result of enhanced CHO oxidation, especially during the later stages of the exercise task. In contrast to this research, Foster *et al.* (1979) found no improvements in performance following CHO ingestion, observing, if anything a decrease in performance following supplementation. In this study, participants were provided with water, 75g of glucose or a liquid meal (10g protein, 12.5g fat, 15g CHO) 30- 45min prior to cycling to exhaustion at 80 and  $100\% \dot{V}O_{2\max}$ . A 19% reduction in endurance performance time at  $80\% \dot{V}O_{2\max}$  was observed following ingestion of the glucose feeding compared to the water trial. The pre- exercise feedings had no effect on exercise time at  $100\% \dot{V}O_{2\max}$ . The observed suppression of FFA throughout the glucose trial led the authors to conclude that glucose feedings in the 30- 45min period before endurance exercise increased the rate of CHO oxidation and impaired the mobilization of FFA, subsequently reducing exercise time to exhaustion ( $P < 0.05$ ). Conflicting with both sides of this debate, is the research of Devlin *et al.* (1986) who observed no significant difference in endurance performance or muscle glycogen depletion following CHO ingestion. In this study, participants were provided with either a snack food or a placebo, 30min before an intermittent cycling exercise at  $70\% \dot{V}O_{2\max}$ . Although the snack food elicited significantly higher plasma glucose ( $28 \pm 9.7 \text{ mg/dl}$ ) and insulin ( $219 \pm 61 \text{ pmol.l}$ ) responses before exercise compared to the placebo ( $0.1 \pm 0.8 \text{ mg/dl}$  and  $-7 \pm -5.5 \text{ pmol.l}$ , respectively,  $P < 0.05$ ), these differences were no longer present by the end of the first exercise period. In addition, no improvements in endurance performance ( $52 \pm$

6.4 and  $48 \pm 5.6$ min) or muscle glycogen depletion ( $56 \pm 14.7$ , and  $50 \pm 15.5$  micrograms.mg protein) following exercise was observed between the snack food and placebo groups, respectively, demonstrating that the ingestion of mixed-macronutrient snacks, 30min prior to exercise does not negatively influence endurance performance, as previously suggested (Foster *et al.*, 1979).

One concern about feeding CHO prior to exercise is that a rapid increase in blood glucose and thus insulin might cause hypoglycaemia at the start of the activity (see Figure 1.2 and 1.3). A second effect of hyperinsulinaemia prior to exercise is a reduction in lipolysis, and fat utilisation (Kirwan *et al.*, 2001). Both of these conditions may accelerate the oxidation of CHO, subsequently increasing reliance on muscle glycogen during the exercise, producing a potential disadvantage to exercise metabolism and performance. These metabolic alterations have been observed even when CHO was consumed four hours before exercise, and persisted despite normalisation of plasma glucose and insulin concentrations at the onset of exercise (Coyle *et al.*, 1985).



**Figure 1.2 and 1.3. Plasma insulin and blood glucose responses, respectively, following ingestion of a meal (Lee and Wolever, 1998).**

The effect of pre- exercise CHO ingestion has remained controversial based primarily on studies showing that ingestion of CHO 30 to 60min before exercise may lead to hyperinsulinaemia and hypoglycaemia and that exercise performance may be subsequently impaired (Foster *et al.*,



1979; Kuipers *et al.*, 1999). It was the previously described study published by Foster *et al.* (1979) that created this initial confusion among athletes and sports scientists by demonstrating that feeding glucose 30min prior to a cycling bout impaired cycle time to exhaustion by 19% at 80%  $\dot{V}O_{2max}$ . The investigators observed that relative to a control trial, blood glucose and FFA concentrations stayed depressed for the glucose trial throughout exercise. Although muscle glycogen content was not determined, the reduction in endurance following CHO feeding was attributed to an accelerated muscle glycogenolysis, an increased rate of CHO utilization and decreased rate of lipid mobilisation after CHO ingestion. From this, Foster *et al.* (1979) concluded that glucose should not be consumed shortly before an event because this practice can cause relative hypoglycaemia and suppression of FFA concentrations in the blood.

Similarly, Costill *et al.* (1977) demonstrated that when FFA concentrations are increased by heparin (anticoagulant), muscle glycogen utilisation is reduced. However, when 75g glucose was ingested 45min before exercise, hyperinsulinaemia coupled with the enhanced insulin sensitivity and decreased hepatic glucose production caused a rapid fall in blood glucose concentrations to a hypoglycaemic state, which continued until the end of exercise and caused a significantly greater rate of glycogen utilisation. These inconsistencies within the literature may be related to a number of factors such as the timing of the meal ingestion or the quantity of CHO ingested. However, another important factor appears to be the rate at which CHO foods are both absorbed and oxidised. Carbohydrate foods, which contain a combination of nutrients, are oxidised and absorbed at different rates and therefore elicit different effects on glycaemia and hyperinsulinaemia (Ludwig, 2002).

### **1.5 Glycaemic Index**

Glucose and fructose are sugars, which are frequently used to examine the effect of pre- exercise CHO ingestion on metabolism and performance. These sugars have different absorption rates and consequently display different effects on levels of circulating hormones and blood- borne substrates (Jentjens and Jeukendrup, 2005). Pre- exercise ingestion of glucose, for example, typically results in



hyperglycaemia followed by a large rise in plasma insulin (Foster *et al.*, 1979). This can result in rebound hypoglycaemia (a swing to a low level of glucose in the blood after having a high level), subsequent lower circulating FFAs, and an increased CHO oxidation and muscle glycogen utilisation and in this case an impaired performance. In contrast, fructose blunts this rise in glucose and insulin as only half of the CHO is absorbed, and much slower than that of glucose (Thomas and Wolever, 2003). As a consequence, glycogenolysis is similar or decreased when compared to the ingestion of a placebo.

One way to counteract this hyperglycaemic/ insulinaemic problem may be to consume a CHO- rich meal that elicits an attenuated blood glucose response curve due to delayed digestion and absorption of the meal. Such meals that elicit these reduced glycaemic responses have been found to have a reduced glycaemic index (GI) (Jenkins *et al.*, 1981). These varying glycaemic and insulinaemic responses following the ingestion of different CHO allows for the classification of CHO that is more informative than describing them as either simple or complex. These simple CHO- rich molecules elicit rapid and short lived raises in blood glucose concentrations, whereas complex CHO- rich foods produced a flatter and sustained rise in blood glucose. The problem with classifying CHO foods as simple (mono- and disaccharides) or complex (polysaccharides) is that this suggests that simple molecules are absorbed more rapidly than more complex, larger molecules. However, digestion and absorption do not always occur at the same rates for all carbohydrates within a specific chemical grouping (Ludwig, 2002). An example of this was previously described with glucose and fructose, although both monosaccharides, fructose is absorbed from the intestine more slowly than glucose. Fructose is absorbed from the intestine by GLUT5 transporters (Burant *et al.*, 1992; Ferraris and Diamond, 1997) and intestinal glucose transport occurs via a sodium-dependent glucose transporter (SGLT) 1 (Ferraris and Diamond, 1997). Therefore Jenkins *et al.* (1981) devised the glycaemic index to provide a more accurate indicator of the glycaemic and insulinaemic responses of individual foods.

The concept of the GI was proposed to evaluate the impact of different sources of CHO on post- prandial blood glucose response using comparisons with a glucose solution or white bread

standard. The index was developed because the blood glucose response to CHO ingestion, as previously discussed, is not a simple function of the total amount of CHO consumed, but is instead related to a complex interaction between several factors that affect digestion and absorption, and the rate at which glucose is delivered to the blood (Kirwan *et al.*, 2001). Therefore, even if the amount of CHO is fixed, the blood glucose response may vary considerably. The GI is calculated by measuring the incremental area under the blood glucose curve following the ingestion of a test food which provides 50g of CHO. This is compared with the area under the blood glucose curve following an equal CHO intake from reference food (Jenkins *et al.*, 1981). The GI typically reflects the overall rate of digestion and absorption of a CHO food, which can be influenced by several factors. These factors include the biochemical structure of the CHO, the absorption process, the size of the food particle, the degree of thermal processing, the contents and timing of the previous meal, and the co- ingestion of fat, fiber and protein (Braaten *et al.*, 1994; Tappy *et al.*, 1996).

In addition to the glycaemic index, more recently, the glycaemic load (GL) was proposed as the product of the GI and the amount of CHO in a serving of food. The GL is calculated by multiplying the GI by the amount of CHO within that particular food. The GL is a recent topic of interest regarding the definition and analysis of the available CHO from the amount consumed in a meal. However, recent research into this observed no significant differences in the GI of two sample foods, despite different glycaemic loads (Granfeldt, Wu, and Bjork, 2006). This supports earlier research (Granfeldt *et al.*, 1995), which also observed no differences in glucose or insulin responses to Arepa meals, despite a 36% difference in available starch intake (29 instead vs. 45g). Similarly, Wolever and Bolognesi (1996) observed identical glucose and insulin responses to boiled barley regardless of whether the participants consumed 50g or 100g. These studies suggest that the glycaemic load may not be so important to the glucose and insulin response, rather, the presence of both dietary fibre and resistant starch may predominantly affect these insulinaemic responses.

The GI provides a way to measure and utilise the individual blood glucose profiles achieved by consuming various CHO- rich foods and drinks. The GI has also been shown to provide a reliable and consistent measure of relative blood glucose response to CHO- rich foods and meals

and has therefore implications for manipulating meals and diets to produce desired metabolic or clinical outcomes. This can be useful for the treatment of diabetes, hyperlipidemias, and potentially obesity (Miller, 1994; Wolever, *et al.*, 1990). However, although the GI was originally devised to help diabetic individuals with the glycaemic control of their diet (Jenkins *et al.*, 1981), the concept now has a wider applicability. Given the information that can be provided by knowing the GI of particular foods, pre- exercise nutritional guidelines on the selection of appropriate carbohydrates to consume before, during and after exercise can be provided to athletes. These nutritional guidelines are employed as a possible strategy to reduce the hyperglycaemia and insulinaemic effects associated with the pre- exercise ingestion of carbohydrates and the subsequent effects these responses can have on exercise performance. The development and use of the GI has queried the appropriateness of generalising results from studies of glucose solutions of purified CHO to predict blood glucose responses to any CHO, particularly those ingested in foods. This is essential as many of the early findings relating dietary CHO to exercise performance did not always differentiate between types of CHO and their food sources. Most of these studies used simple sugars, often in solution, which would elicit glycaemic responses different from starch, especially if starch was consumed in solid form (Jenkins *et al.*, 1981; Wolever *et al.*, 1986). Therefore the GI has been extensively used in sports nutrition to provide effective information for the planning of appropriate nutritional strategies on CHO supplementation prior to exercise (Burke *et al.*, 1999; Hargreaves, 1991; Walton and Rhodes, 1997).

### **1.6 Mechanisms of Action:**

The pancreas contains both exocrine and endocrine tissues, of which the endocrine tissue is responsible for the production and secretion of hormones into the bloodstream, most importantly insulin and glucagon. Insulin, which is synthesised within the  $\beta$ - cells, is secreted according to the metabolic or nutritional state of the individual, but is predominantly regulated by the concentration of glucose within the plasma (Frayn, 1996). The response to an increase in glucose concentration is

an increase in the rate of glucose metabolism, leading to the production of ATP (Wilson *et al.*, 1998).

The homeostatic feature of blood glucose is brought about by coordinated control of various aspects of glucose metabolism, of which insulin plays a major role (Huang and Czech, 2007). There is a characteristic dose- response curve for insulin secretion rate against glucose concentrations as insulin secretion is not increased much until the glucose concentration rises above  $5\text{mmol}\cdot\text{l}^{-1}$ , which is the normal concentration of glucose in the plasma (Wilson *et al.*, 1998). Contrasting with the effects of insulin to glucose is the polypeptide known as glucagon, which is secreted from the pancreatic  $\alpha$ - cells and acts to elevate the blood glucose concentration (Frayn, 1996). However, unlike insulin, glucagon secretion is suppressed by a rise in blood glucose concentration and therefore, a decrease in the plasma glucose concentration will lead to an increased ratio of glucagon to insulin (Febbraio *et al.*, 2000).

In the post- absorptive state, the blood glucose concentration is usually a little under  $5\text{mmol}\cdot\text{l}^{-1}$ . The concentration of insulin in plasma varies widely between individuals, but is typically approximately  $60\text{pmol}\cdot\text{l}^{-1}$ . The rate of turnover of glucose in the post- absorptive state is close to  $2\text{mg}\cdot\text{kgbw}^{-1}\cdot\text{min}$  of glucose, or 130mg of glucose every min entering and leaving the circulation (Wilson *et al.*, 1998). In this post- absorptive state, glucose, from either hepatic glycogenolysis or gluconeogenesis, enters the blood almost completely from the liver. Depending on factors such as diet and exercise, approximately two- thirds of the glucose is from glycogen breakdown ( $80\text{mg}\cdot\text{min}^{-1}$ ). The remainder of glucose entry ( $50\text{mg}\cdot\text{min}$ ) will result from gluconeogenesis, of which lactate will constitute a little over half, and alanine (largely from muscle) and glycerol (from adipose tissue lipolysis) will contribute to most of the remainder (Febbraio *et al.*, 2000).

The first meal of the day provides the largest switch from production to storage, the CHO of which is digested and absorbed from the intestine (Frayn, 1996). Any increase in glucose concentration is typically detected within about 15min, and this increase continues for 30- 60min after a moderate breakfast. The exact timing of detection depends on factors including timing of the

meal, glycaemic index and fibre content (Ludwig, 2002). As the concentration of blood glucose rises, the endocrine tissues of the pancreas respond by stimulating insulin secretion. The liver receives the blood draining the small intestine in the hepatic portal vein, which leads to an inflow of glucose into hepatocytes via the transport protein known as GLUT 2. The elevation of intracellular glucose concentration in hepatocytes, together with the change in the insulin to glucagon ratio, leads to inactivation of glycogen phosphorylase and activation of glycogen synthase (Wilson *et al.*, 1998).

There is always an elevation of the blood lactate concentration after ingestion of CHO, therefore, the overall effect is that some of the glucose arriving in the blood will be used by tissues, released into the blood as lactate, and then taken up by the liver and converted to glucose- 6-phosphate and subsequently glycogen, which is the indirect pathway of glycogen deposition (Salway, 1995). This gluconeogenic flux does not lead to the release of glucose into the blood as glucose- 6- phosphate but is instead directed into glycogen synthesis. Therefore, the direction of lactate into glycogen in the liver can be seen as part of an intense drive to store as much as possible of the incoming glucose, even if it supplies some energy to other tissues first (Frayn, 1996).

The rate of glucose release from hepatocytes (i.e. release of glucose from glycogen and gluconeogenesis) falls dramatically, almost to zero, within 1-2 h after a glucose load or CHO meal. At the same time hepatocytes will take up glucose arriving in the portal vein. Total glucose release into the circulation through the hepatic veins, however, increases because of exogenous (dietary) glucose coming from the small intestine (Huang and Czech, 2007).

Other tissues respond to the increase in insulin concentration. The major cellular mechanism for exogenous glucose disposal is insulin- stimulated glucose transport into skeletal muscle. In skeletal muscle and adipose tissue, glucose uptake is stimulated by the rise in insulin through both the increased activity of the transport proteins GLUT1 and GLUT4 at the cell membrane, and by an increased disposal of glucose within the cell (Huang and Czech, 2007). GLUT4 is highly expressed in adipose tissue and skeletal muscle and is the principle glucose transporter that mediates this insulin- stimulated glucose transport into the skeletal muscle. At the same time the plasma concentration of non- esterified fatty acids falls because fat mobilisation in adipose tissue is

suppressed. Therefore, tissues such as skeletal muscle, which can use either fatty acids or glucose as their energy source, switch to utilisation of glucose. The GLUT4 glucose transporter is therefore a major mediator of glucose removal from the circulation and a key regulator of whole body glucose homeostasis.

Not all the glucose taken up by the muscle is oxidised under these conditions: insulin also activates muscle glycogen synthase, and glycogen storage will replenish muscle glycogen stores. Therefore, after a CHO - containing meal there is a general switch in metabolism to the use of glucose rather than fatty acids, but there is also a major switch to the storage of glucose as glycogen (Wilson *et al.*, 1998).

### **1.7 Disposal of glucose after a meal:**

The amount of glucose in a meal (typically 80-100g) would be enough to raise the concentration of glucose in the plasma about eight- fold. In a normal healthy person, the peak glucose concentration after breakfast will be approximately  $7\text{-}8\text{mmol}\cdot\text{l}^{-1}$ , a rise of only 60% at most from the post absorptive value of  $5\text{mmol}\cdot\text{l}^{-1}$  (Huang and Czech, 2007). The change in insulin concentration will be much greater rising from  $\sim 60\text{pmol}\cdot\text{l}^{-1}$  to approximately  $400\text{-}500\text{pmol}\cdot\text{l}^{-1}$ . The glucagon concentration in systemic (mixed) blood may not change much, but there will be a change in the insulin to glucagon ratio. By the end of the absorptive state- about 5h after the meal, approximately 25g of the 100g of CHO ingested will have been stored, and 75g oxidised, which indicates that, although glucose oxidation in tissue is increased after a meal, the drive for glucose storage is such that around one- quarter of the glucose is stored for later use (Frayn, 1996).

### **1.8 Metabolic Effects of Exercise:**

The sympathetic nervous system and adrenaline bring about the mobilization of stored fuels. The plasma glucose concentration may rise or fall during exercise depending largely on the nutritional state of the athlete (Romijn *et al.*, 1993). Insulin concentration falls somewhat during endurance exercise, which represents  $\alpha$ - adrenergic inhibition of its secretion from the pancreas, brought about

by the increased concentration of circulating adrenaline. Therefore, the glucagon to insulin ratio reaching the liver will undoubtedly rise, which favours the breakdown of glycogen (Ludwig, 2002).

With regards to the effects on exercise performance, the availability of CHO for exercise can potentially be optimised by manipulating the GI of the CHO supplementation. A low GI, CHO-rich meal has been recommended as the CHO supplement before prolonged exercise in order to promote the availability of the sustained CHO (Thomas *et al.*, 1991; 1994; Wee *et al.*, 1999; Wu *et al.*, 2003; Sparks *et al.*, 1998; DeMarco *et al.*, 1999; Febbraio and Stewart, 1996., Febbraio *et al.*, 1996; 2000). Whereas, a high GI, CHO- rich meal appears to be beneficial for glycogen storage post- exercise by promoting greater glucose and insulin responses and being absorbed and digested at a faster rate than LGI foods (Stevenson *et al.*, 2005; Wee *et al.*, 2005). Another attraction of consuming low rather than high GI CHO foods before exercise is that the normal suppression of fatty acid mobilization associated with hyperinsulinemia is less and so there is a greater contribution of fat to energy metabolism at the expense of CHO oxidation. Both these factors will have important implications for both endurance- trained athletes and those who are aiming to reduce body fat mass for health purposes (Wu *et al.*, 2003).

Despite these observed metabolic responses, even when the pre- exercise meal is quantified according to the GI, the literature regarding both fuel availability and contribution is inconsistent. Some (Sparks *et al.*, 1998; Thomas *et al.*, 1991; 1994; Wee *et al.*, 1999) but not all (Febbraio and Stewart, 1996; Horowitz and Coyle, 1993; Kirwan *et al.*, 1998) studies observed the ingestion of a pre- exercise LGI meal to reduce CHO oxidation when compared to the ingestion of a HGI meal. Of note, only two of these studies sampled muscle tissue and found the rate of glycogenolysis to be unaffected by GI, which is not surprising because these same studies observed similar rates of CHO oxidation during exercise (Febbraio and Stewart, 1996; Kirwan *et al.*, 1998). Only a few studies (Burke *et al.*, 1993; Febbraio and Stewart, 1996; Kirwan *et al.*, 1998; 2001; Sparks *et al.*, 1998; Stevenson *et al.*, 2005) have investigated the effects of feeding different forms of CHO on factors related to exercise metabolism (blood glucose, FFA, insulin, respiratory exchange ratio (RER), muscle glycogen use) or to performance (ratings of perceived exertion (RPE), time to exhaustion).

Therefore, clearly the hypothesis that pre- exercise ingestion of LGI food would be advantageous both during exercise and to performance, by reducing CHO oxidation, requires further examination.

### **1.9 Effects of the GI on Exercise Performance:**

Several studies have examined the effect of the GI of foods on metabolism and exercise performance consumed at different times prior to the prolonged exercise. Early studies of pre-exercise CHO feedings showed different metabolic outcomes according to the type of sugar ingested. In particular, studies showed that fructose, a low- GI saccharide, could be consumed before exercise without producing the metabolic impairments seen with glucose feedings (Hargreaves *et al.*, 1985). However, Thomas *et al.* (1991) were the first to suggest a potential role for the GI in the area of sports nutrition by undertaking a manipulation of the glycaemic response to pre- exercise CHO- rich meals. In this study, eight trained cyclists consumed a meal providing one gram of CHO per kilogram body mass in the form of a low- GI food (lentils, GI = 29), one hour prior to cycling at 67%  $\dot{V}O_{2\max}$ . Time to exhaustion was significantly prolonged by 20min (~ 20%) in the LGI trial compared with the ingestion of an equal amount of CHO eaten in the form of a HGI food (potatoes, GI = 70), drinking a glucose solution or water ( $P < 0.05$ ). The exercise times for the lentils, potatoes, glucose and water were 117, 98, 108, and 99min respectively. There was a significant difference in exercise time for the lentils trial, but there was no significant difference in performance between the potato, glucose, or water trials. These results were attributed to attenuated glycaemic and insulinaemic responses before exercise in the LGI trial compared with the HGI meal, maintaining more plasma glucose levels during exercise and increased FFA concentrations ( $P < 0.05$ ). Reduced values for the respiratory exchange ratio (RER) were observed throughout the LGI trial ( $P < 0.05$ ), indicating reduced use of CHO to fat. Although muscle glycogen was not measured, the authors suggested that glycogen sparing may have occurred with the LGI trial (Thomas, *et al.*, 1991). This study offers support for the consumption of LGI foods prior to exercise; however, it did not address the different rates of digestion and absorption of the three CHO foods within the hour before the start of exercise. In addition, the foods were not iso-



macronutrient, in that only the amount of CHO ingested in these trials had been matched, and the total energy content of the LGI lentil meal was actually 36% higher than the HGI potato meal. These study limitations may have impacted on the glycaemic responses of the meals as the addition of fat and protein has been found to reduce the glycaemic responses of the CHO sources (Horowitz and Coyle, 1993), increase plasma FFA concentration (Burke *et al.*, 1995) and delay gastric emptying (Estrich *et al.*, 1967), all of which will have had an impact on metabolic responses and thus the interpretation of these results. Nonetheless, this study revived the prejudices about the metabolic perturbations caused by CHO feedings prior to exercise, and suggested an alternative way to increase or sustain CHO availability during exercise while attenuating any negative responses of pre- exercise feedings. The results of the Thomas study have been widely publicised and are largely responsible for the general advice that athletes should choose pre- exercise meals based on LGI CHO- rich foods and drinks.

Similar findings were corroborated in 1999 by DeMarco *et al.*, (1999) who demonstrated that a LGI meal consumed 30min before exercise can exhibit a beneficial effect on maximal performance following sustained exercise. In this particular study, ten trained cyclists performed an exercise task of 2h cycling at 70%  $\dot{V}O_{2\max}$  followed by cycling to exhaustion at 100%  $\dot{V}O_{2\max}$ , as a means to measure their subsequent maximal effort. Either a moderately- high GI meal or LGI meal was consumed 30min prior to the exercise task. The amount of CHO provided in both meals was equal to 1.5g.kgbw<sup>-1</sup>. The exhaustion time was significantly prolonged by 59% in the LGI trial (206.5 ± 43.55s) compared with the HGI trial (129.5 ± 22.85s) (P<0.05). It was also found that the plasma insulin levels were significantly lower after the LGI meal than after the HGI meal through 20min exercises (P<0.05). After the LGI meal, plasma glucose levels were higher (P<0.05), RPE was lower and the RER values were lower until 2h of exercise when compared with the HGI trial (P<0.05). The higher fat oxidation and possibly an increase in the availability of FFA's as an energy source for exercise were supported by the lower RER values. The investigators concluded that a pre- exercise LGI meal may positively affect the maximal performance following sustained exercise. The improvements in the subsequent maximal effort is supported by the maintenance of

the higher plasma glucose levels at the end of 2h strenuous exercise following the consumption of the LGI meal, when results were compared to that of the HGI meal.

Additional research in this area by Kirwan *et al.* (1998; 2001) offered support for the performance enhancing effects of LGI foods in two separate studies. In the first study, six recreationally active women consumed one of three test meals 45min prior to cycling to exhaustion, at 60%  $\text{VO}_{2\text{peak}}$ . The test meals containing 75g glucose consisted of sweetened whole- grain rolled oats or sweetened whole- oat flour. Although the diets were matched for macronutrient content, the whole- grain rolled oats meal contained greater dietary fiber content than the whole- oat flour meal (6.1 vs. 3.1g, respectively). These test foods provided a GI of ~60- 70. Kirwan *et al.* (1998) concluded that eating a moderate GI (~63) breakfast cereal (whole- grain rolled oats) with a high dietary fiber content, 45min before exercise resulted in a 41min (16%) increase in time to exhaustion during prolonged sub maximal exercise ( $P<0.05$ ). Kirwan *et al.* (1998) suggested that the improved performance may be due to an attenuated insulin response and a concomitant reduction in the suppression of FFA ( $P<0.05$ ), possibly sparing endogenous glucose, producing a glucose- sparing effect that facilitated sustained energy production.

More recently, Kirwan *et al.* (2001) used a similar protocol to investigate the effects of the GI on exercise performance and metabolism. In this study, six apparently healthy, active male participants consumed either a MOD-GI meal (whole- grain rolled oats, GI = ~61) or a HGI meal (puffed rice, GI = 82), each providing 75g of available CHO 45min before being asked to cycle to exhaustion at 60%  $\text{VO}_{2\text{peak}}$ . Following this protocol, exercise performance times were significantly longer ( $P<0.05$ ) after the MOD-GI meal (165min  $\pm$  11) compared to the HGI meal (141min  $\pm$  8). It was found that the ingestion of the MOD-GI breakfast cereal was associated with greater plasma glucose levels ( $P<0.05$ ) at 60 ( $5.2 \pm 0.1\text{mmol}\cdot\text{l}^{-1}$ ) and 90 ( $4.8 \pm 0.1\text{mmol}\cdot\text{l}^{-1}$ ) min after the meals compared to the HGI meal ( $4.2 \pm 0.2\text{mmol}\cdot\text{l}^{-1}$  and  $4.1 \pm 0.1\text{mmol}\cdot\text{l}^{-1}$ ). Carbohydrate oxidation was also significantly greater ( $P<0.05$ ) during the MOD-GI trial and was directly correlated with exercise performance time ( $r = .95$ ,  $P<.0001$ ). These observations led the authors to suggest that this 23% increase in performance was due to the higher level of glycaemia and greater total CHO

oxidation after the MOD-GI meal. Additionally, total CHO oxidation was significantly correlated with exercise performance, suggesting that improvements in performance are dependant on whether the meal can maintain adequate euglycemia during exercise to sustain CHO oxidation. Interestingly, as muscle glycogen use did not differ between trials, it is most likely that the additional CHO oxidized during the MOD-GI trial came from the ingested meal. In support of this observation, the estimated difference in CHO use between the MOD-GI and the water control was 75g, which is exactly the amount of CHO that was provided in the meal. Thus it seems reasonable to suggest that during the MOD-GI trial, the meal provided adequate glucose for oxidative metabolism and may have also reduced the dependence on hepatic glycogenolysis during the early phase of exercise. The results of this study suggest that individuals who perform exercise or physically demanding work for prolonged periods of time may benefit from eating a MOD-GI meal before the activity. What does need to be considered with this and the above study, however, is the intensity of  $\dot{V}O_{2peak}$  at which the participants were working at. The participants were required to work at  $60\%\dot{V}O_{2peak}$ , which, as already discussed can be supported predominantly by the oxidation of fat (Romijn *et al.*, 1993; Stisen *et al.*, 2006). Therefore, this intensity may not have challenged the glycogen stores enough to determine if the test meals actually enhanced endurance capacity and may also explain why there was no difference in muscle glycogen utilisation between the trials as they were not being challenged enough.

In a more recent study, Wu *et al.* (2003) examined the influence of the GI on substrate utilisation. This study provided nine healthy male recreational runners ( $\dot{V}O_{2max}$  58.1ml.kg.min<sup>-1</sup>) with one of three isoenergetic test meals. Both the LGI meal (GI = 36.9: bran flakes, skimmed milk, peaches, apples and apple juice), and the HGI meal (GI = 77.4: corn flakes, skimmed milk, white bread, jam, high CHO drink) provided 2g.kgbw<sup>-1</sup> of carbohydrate and were consumed 3h before a 60min treadmill run at  $65\% \dot{V}O_{2max}$ . Analysis of the data revealed that the HGI meal was associated with greater glycaemic and insulinaemic responses during the postprandial period compared with the LGI trial ( $P < 0.05$ ). Although muscle glycogen concentration was not measured in the present study, the authors suggested that this higher ( $P < 0.05$ ) fat oxidation in the LGI meal

may spare muscle glycogen during exercise, potentially improving endurance performance by delaying the depletion of muscle glycogen. However, exercise performance in this study was not assessed, although the metabolic perturbations in this study do corroborate those of previous research, where a beneficial effect on performance was observed (Thomas *et al.*, 1991; Kirwan *et al.*, 1998; 2001).

Offering further support for the beneficial effect of LGI meals on exercise performance are two separate studies by Stevenson *et al.* (2005) and Wu and Williams (2006). In the former study, nine male recreational athletes were provided with HGI (GI = 70) or LGI (GI = 35) recovery diets following glycogen depleting exercise, which provided  $8\text{g.kg}^{-1}$  of CHO over the next 22h. The next day, in the fasted state, the participants were required to run to exhaustion at  $70\% \dot{V}O_{2\text{max}}$ . Time to exhaustion on the LGI recovery diet was significantly longer ( $108.9 \pm 7.4\text{min}$ ,  $P < 0.05$ ) compared to the HGI trial ( $96.9 \pm 4.8\text{min}$ ). These results corroborate those of earlier studies by demonstrating an improvement in performance following the ingestion of an LGI recovery meal (Thomas *et al.*, 1991; DeMarco *et al.*, 1999; Kirwan *et al.*, 1998; 2001). Higher free fatty acid concentrations and fat oxidation rates were also observed for the LGI trial ( $P < 0.05$ ), again supporting previously observed metabolic perturbations associated with the consumption of LGI foods. The authors, similarly to other proposed explanations (Kirwan *et al.*, 1998; 2001), postulated that this increase in endurance capacity may be due to the higher rates of fat oxidation in the LGI trial, again producing a possible glycogen sparing effect. However, the authors suggested that these higher rates of fat oxidation may be the result of lower pre-exercise muscle glycogen concentrations compared to the HGI trial, as pre-exercise substrate availability has emerged as an important regulator of the patterns of fuel oxidation during exercise (Arkinstall *et al.*, 2004). The applicability of these results also need to be interpreted with caution as the exercise protocol employed in this study is not representative of normal dietary practice, in that participants were required to run to exhaustion in the fasted state following an overnight fast. Nevertheless, consumption of the LGI recovery meal significantly enhanced endurance capacity compared to ingestion of an isocaloric, nutrient matched HGI recovery meal.

In a more recent study by Wu and Williams (2006), eight male recreational runners were provided with isocaloric LGI (GI = 37) or HGI (GI = 77) meals, each providing  $2\text{g.kgbw}^{-1}$  of CHO. These meals were consumed 3h prior to performing running to exhaustion at  $70\% \dot{V}O_{2\text{max}}$ . Consistent with other research, fat oxidation rates and plasma glucose concentrations were higher and more stable following the LGI meal and time to exhaustion was also significantly longer in the LGI trial ( $108 \pm 4.1\text{min}$ ) than in the HGI trial ( $101.4 \pm 5.2\text{min}$ ) ( $p = 0.038$ ). Similarly, fat oxidation rates were significantly higher following the ingestion of an LGI meal ( $P < 0.05$ ). This and some of the aforementioned research are some of the latest in this area demonstrating favourable changes in exercise metabolism, if not exercise performance following the ingestion of LGI foods and meals.

Although these above studies demonstrate improvements in exercise performance following consumption of LGI foods before exercise (DeMarco *et al.*, 1999; Kirwan *et al.* 1998;2001; Stevenson *et al.*, 2005; Thomas *et al.*, 1991; Wu and Williams, 2006), there still remains a lot of controversy in this area with some studies demonstrating no significant effect of the GI on exercise performance (Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Stannard *et al.*, 2000; Thomas *et al.*, 1994; Wee *et al.*, 1999). Thomas *et al.* (1994) conducted a study that repeated the protocol of their original research in 1991, where six trained cyclists consumed one of the four test- foods 1h prior to cycling to exhaustion at  $65\text{-}70\% \dot{V}O_{2\text{max}}$ . The four test meals had a GI of 30, 36, 73 and 100 respectively and provided  $1\text{g.kgbw}^{-1}$  of CHO. A negative correlation was observed between the GI of a meal and the depression of blood glucose and FFA concentration during exercise, with the LGI meals being associated with higher glucose and FFA concentrations than the HGI meals after 90min of exercise. An increase in performance would therefore be expected, however, in this study there was no significant difference in time to exhaustion between trials (mean  $\pm$  SEM =  $95 \pm 12\text{min}$ ) test meals. The results suggested that the slow digestion of CHO in the meal prior to exercises favours the higher concentration of energy in the blood toward the end of exercises. However, it should be noted that although the CHO content of the test meals was matched ( $1\text{g.kg}$ ), the diet was not matched for protein, fat or total energy content, which ranged from 308 to 490kcal (1271 to 2024kJ) per meal. Therefore these data should be interpreted with caution.

Similar findings were obtained through Febbraio and Stewart (1996), who examined the effects of the GI of a pre- exercise CHO meal on muscle metabolism and prolonged exercise performance. In this study, six endurance trained men consumed either a HGI meal (instant mash potato, GI = 80), a LGI meal (lentils, GI = 29), both providing  $1\text{g.kg}^{-1}$  of CHO, or a placebo meal (diet jelly) 45min before an exercise trial. During the exercise trial, participants cycled at a workload corresponding to 70%  $\dot{V}O_{2\text{max}}$  for 120min, followed by a 15min performance cycle where the total work (kJ) was measured. The results obtained revealed that the ingestion of the HGI food significantly elevated ( $P < 0.01$ ) blood glucose concentration 15min after ingestion, compared to both the LGI and control trial, but no further differences in glycaemic responses were observed to the meals thereafter, despite the plasma insulin concentration being greater ( $p < 0.01$ ) in the HGI trial throughout the rest period after ingestion. Although significantly lower FFA concentrations were observed throughout exercise in the HGI trial compared with LGI, there was no difference in CHO oxidation or muscle glycogen concentration ( $\sim 240\text{mmol.kg}^{-1}$ ) during the submaximal cycling and total work outputs during and post, respectively, the performance cycle across all trials. A similar amount of total work performed during the performance cycle was observed across all trials ( $\sim 250\text{kJ}$ ). These observations lead the authors to conclude that the pre- exercise CHO ingestion, while it increases oxidation, it does not influence the rate of muscle glycogen utilisation or exercise performance, irrespective of the glycaemic and insulinaemic responses to meals with varying GI's, pre- exercise ingestion of CHO has no effect on muscle glycogen utilization or exercise performance.

Similarly, Sparks *et al.* (1998) investigated the effect of GI of CHO ingestion 45min before exercise on exercise metabolism and performance. Eight endurance- trained triathletes ingested a HGI meal (instant mashed potato, GI = 80), LGI meal (lentils, GI = 29), or a placebo meal (low energy soft drink) 45min prior to an exercise task. The amount of CHO ingested was equal to  $1.0\text{g.kg}^{-1}$  CHO. The exercise task consisted of a 50min cycle at 67%  $\dot{V}O_{2\text{max}}$  and then a subsequent 15min self- paced performance trial in which the total work was measured. The metabolic responses observed were consistent with the GI of the foods, in that the HGI meal was

associated with significantly higher plasma glucose concentrations 30min postprandial compared to the LGI meal. This declined at the onset of exercise and was lower ( $P<0.01$ ) at both 10 and 30min after exercise than the LGI concentration. Plasma insulin concentrations were significantly higher at rest following ingestion and at all time points during exercise in the HGI trial compared to the LGI trial. Plasma free fatty acid concentrations were significantly higher in the LGI trial at the onset and termination of exercise compared to the HGI trial. Both CHO oxidation rates and RER were significantly higher for the HGI trial than the LGI trial during submaximal exercise. Despite these favourable metabolic perturbations no significant differences in total work produced during the performance ride was observed between the trials ( $249 \pm 12$ ;  $253 \pm 10$ ;  $254 \pm 12$ kJ for HGI, LGI, and CON, respectively). These data suggests that despite possible favourable alterations in metabolism, there appears to be no apparent ergogenic effect of this on exercise performance.

Wee *et al.* (1999) conducted a study to examine the influence of high and low GI meals on endurance running capacity. They found that the GI of the meal consumed 3- hour prior to exercise does not influence the subsequent endurance capacity. Eight active runners consumed either a HGI (potatoes, tuna, sweet corn, crumpets, and honey) meal or a LGI (boiled red lentils) meal 3h before a run to exhaustion at  $70\% \dot{V}O_{2\max}$ . The test meals both provided  $2.0\text{g.kgbw}^{-1}$  of CHO and were both isocaloric and closely matched for macronutrient content ( $846 \pm 21\text{kcal}$ ,  $144.4 \pm 3.6\text{g CHO}$ ,  $4.1 \pm 0.1\text{g fat}$  and  $64.3 \pm 1.6\text{g protein}$  (HGI); and  $852 \pm 21\text{kcal}$ ,  $144.4 \pm 3.6\text{g CHO}$ ,  $3.6 \pm 0.1\text{g fat}$ , and  $65.0 \pm 1.6\text{g protein}$  (LGI). Blood glucose values peaked at 15min ( $P<0.01$ ) after ingestion of the HGI meal, whereas, blood glucose did not change significantly for the LGI trial. The incremental areas under the blood glucose curve for 1, 2, and 3h after the HGI meal were 14, 7, and 5- fold greater, respectively than those after the LGI meal ( $P<0.001$ ). Throughout the postprandial period, serum insulin concentrations were significantly higher during the HGI trial than the LGI trial. However at the onset of exercise, the difference was not statistically significant. The overall 3h incremental area under the insulin curve for the HGI trial exceeded that of the LGI trial by 336%. Both serum FFA and glycerol concentrations were significantly higher during the postprandial period following ingestion of the LGI meal compared to the HGI trial. Insulin concentrations did

not differ between trials during exercise, but the blood glucose concentration at 20min into exercise in the HGI trial was lower than the LGI trial at the same time point ( $3.6 \pm 0.3\text{mmol}\cdot\text{l}^{-1}$  vs.  $4.3 \pm 0.3\text{mmol}\cdot\text{l}^{-1}$ , respectively). After 2min, blood glucose concentrations were not different between the two trials. During exercise it was observed that both plasma glycerol and serum free fatty acid concentrations were lower in the HGI trial compared to the LGI trial. During the first 80min of exercise in the LGI trial, CHO oxidation was 12% lower (HGI:  $232 \pm 18\text{g}$  vs. LGI:  $205 \pm 20\text{g}$ ) and fat oxidation was 118% higher than those in the HGI trial (HGI:  $9 \pm 2\text{g}$  vs. LGI:  $19 \pm 3\text{g}$ ). Overall, the total amount of CHO utilized during exercise was higher during the HGI trial ( $308 \pm 22\text{g}$ ) than the LGI trial ( $269 \pm 16\text{g}$ ) ( $P < 0.01$ ). Despite these increased fat oxidation rates associated with the LGI foods, no difference in the run to exhaustion times was observed between trials (LGI:  $111 \pm 6\text{min}$ ; and HGI:  $113 \pm 4\text{min}$ ). The investigators found that although the consumption of a LGI meal 3h before exercise induces a relative shift in substrate utilisation from CHO to fat when compared to a HGI meal, this has no influence on endurance capacity.

Recently, Stannard *et al.* (2000) investigated the effect of the GI of meals consumed 65min before incremental high intensity exercise on exercise performance. Ten trained cyclists performed an incremental cycling ride to exhaustion (load was increased by 50W every 3min with an initial loading of 50W), 65min after the consumption of equal CHO portions ( $1\text{g}\cdot\text{kgbw}^{-1}$ ) of glucose (HGI), pasta (LGI) or a non-CHO placebo (PL). No significant difference was found in the time to exhaustion between trials ( $p = 0.05$ ). In the HGI trial, plasma glucose concentration was significantly lower from 200W until exhaustion when compared to LGI meal. Plasma lactate concentrations after the consumption of HGI meals was higher than consumption of placebo from 30min of rest post-prandial through the end of the 200W workload and from 45min of rest post-prandial through to the end of the 100W workload. The results suggested that the consumption of the HGI meal, 65min prior to the exercise decreases plasma glucose and increases plasma lactate levels during exercise when compared to LGI meal. However, these metabolic responses are not sufficient to produce a detrimental effect on the incremental exercise performance.



### 1.10 GI and glycogen resynthesis

The ability to perform prolonged, exhaustive exercise is closely related to muscle glycogen stores (Bergstrom *et al.*, 1967; Coyle *et al.*, 1986). The reliance on muscle glycogen increases with increasing exercise intensity and a direct relationship between fatigue and depletion of muscle glycogen has been observed (Bergstrom *et al.*, 1967). Furthermore, the initial muscle glycogen concentration has been shown to be related to aerobic endurance performance, and a high exercise intensity cannot be maintained once muscle glycogen stores are depleted (Bergstrom *et al.*, 1967; Hermansen *et al.*, 1967). This research suggests that it is important for performance and most likely for effective training to have significant muscle glycogen stores.

Depletion of body CHO stores is a major cause of fatigue during prolonged exercise, so both pre- and post- event nutritional strategies should focus on optimizing CHO stores in the muscle and liver. The resting values for muscle glycogen in trained muscle are 100- 120mmol.kgww<sup>-1</sup> (Burke *et al.*, 2003), and this will fall to a greater or lesser extent during each training session depending primarily on the duration and intensity of the session. With athletes training or competing on consecutive days, the rapid restoration of muscle glycogen stores during the recovery period is essential as success is often determined by the athlete's ability to recover between one exercise/ training bout and the next. In cycling, the schedule may consist of one or more lengthy events each day, with the competition extending for 1-3 weeks and athletes must train between competitions to maintain fitness. Perhaps even more importantly, the daily training program for many sports, even at a recreational level, involves one or more workouts, with less than 24h separating each session. The ability to recover between training sessions and to maintain quality training without succumbing to the effects of cumulative fatigue and injury is essential in successful athletic performance.

There is a strong drive to replenish glycogen levels in a muscle whose CHO stores have been depleted during exercise, with muscle glycogen storage taking priority over the restoration of liver glycogen content. The rate of synthesis is determined by factors that regulate glucose transport into the cell (for example, glucose supply, and glucose uptake) as well as glucose disposal (for example,

the activity of glycogen synthase enzyme). The intake of CHO after exercise has repeatedly been shown to increase muscle glycogen repletion ( $7.7\text{mmol.kg ww.h}^{-1}$ ) (Tsintzas *et al.*, 2003; Blom *et al.*, 1987; Ivy, 1998). However, in the absence of dietary CHO, synthesis occurs at a low rate ( $1\text{-}2\text{mmol.kgww.h}^{-1}$ ), with some of the substrate being provided through gluconeogenesis. Glycogen storage follows a two- phase timescale: a very rapid synthesis during the first hour of exercise, followed by a slower restoration phase that is under the influence of factors such as insulin. When optimal amounts of CHO are consumed, maximal rates of post- exercise muscle glycogen storage during the first 12h of recovery are within the range of  $5\text{-}10\text{mmol.kgww.h}^{-1}$  and gradually decline as restoration continues. In the face of substantial glycogen depletion, 20- 24h of well- fed recovery are required for the restoration of muscle glycogen stores. Therefore, the consumption of high CHO foods or drinks after exercise is now common practice as muscle glycogen resynthesis is largely dependant on the post- exercise CHO supply.

Glucose uptake by the skeletal muscle is facilitated mainly via the GLUT- 4 transporter. Stimulation of glucose transport by insulin is mediated by translocation of GLUT- 4 from the intracellular sites to the plasma membrane (Lund *et al.*, 1993). Skeletal muscle translocation of GLUT- 4 to the plasma membrane appears to be also facilitated by muscle contraction independent of insulin. It is suggested that, during the initial phase of recovery from exercise, GLUT- 4 still resides in the plasma membrane because of contraction- mediated GLUT- 4 translocation during the exercise bout, and thus glucose can be transported independently of insulin (Hansen *et al.*, 1998; Ivy *et al.*, 1998). After the relatively short period of insulin independent muscle glucose uptake, glucose uptake and GLUT- 4 translocation to the plasma membrane becomes insulin dependant. However, during recovery from exercise, there is marked increase in the sensitivity to insulin of muscle glucose uptake and glycogen synthesis (Price *et al.*, 1994; Richter *et al.*, 1984; Wallberg-Henriksson, 1987). Recent evidence has been presented suggesting that the rate of muscle glucose uptake may control the rate of glycogen resynthesis after exercise. Research from van Hall *et al.* (2000) supports this mechanism in that glucose uptake was highest during the initial recovery period, and leg glucose uptake decreased with the longer time of recovery. GLUT- 4 transporters

that remained in the plasma membrane from the exercise bout may have caused the higher glucose uptake at the initial stage. As the recovery period increases it becomes more dependent on insulin to maintain GLUT- 4 transporters in the plasma membrane and/ or to translocate GLUT- 4 to the plasma membrane. Because the insulin level was low in the water trial, the number of GLUT- 4 transporters in the plasma membrane declined and, therefore, glucose uptake ceased.

Skeletal muscle glycogen resynthesis after exercise has been shown to be biphasic (Maehlum *et al.*, 1978; Price *et al.*, 1994); there is the initial insulin- independent phase of 30- 60min with a high glycogen resynthesis rate followed by an insulin- dependant phase with a slower glycogen resynthesis rate. When Price *et al.* (1994) studied glycogen resynthesis in insulin- resistant participants and controls they did not observe a difference in glycogen resynthesis rate during the insulin- independent phase. However, during the insulin- dependent phase, glycogen resynthesis rate was lower in the insulin- resistant participants (Price *et al.*, 1996). These findings clearly indicate that from 1h of recovery onward insulin might play an important role in the regulation of muscle glycogen resynthesis.

#### *1.10.1 Amount of carbohydrate:*

The most important dietary factor affecting muscle glycogen storage is the amount of CHO consumed. There is a direct relationship between the quantity of dietary CHO and the rate of post-exercise glycogen storage, at least until the muscle glycogen storage capacity or threshold has been reached. Studies suggest that the threshold varies between individuals athletes but generally occurs within the intake of 7-10g·kgbw<sup>-1</sup> (Tsintzas *et al.*, 2003; Blom *et al.*, 1987; Ivy, 1998). Maximal refuelling during the first 0-6h of recovery is achieved when food or drink providing a CHO intake of about 1g.kg<sup>-1</sup> are consumed during the first hour, and repeated every 2h until the normal meal pattern can be resumed by the athlete.

The highest sustained rates of glycogen storage occur during the first few hours after exercise. In addition to the activation of the glycogen synthase enzyme, early post exercise recovery is marked by an exercise- induced permeability of the muscle cell membrane to glucose and an

increase in muscle sensitivity to insulin. CHO feedings during these early stages appear to accentuate these effects by increasing blood glucose and insulin concentrations. The intake of CHO immediately after prolonged exercise has been shown to result in higher rates of glycogen storage ( $7.7\text{mmol.kg ww.h}^{-1}$ ) during the first 2h of recovery, slowing thereafter to more typical rates of glycogen storage ( $\sim 4.3\text{mmol.kg ww.h}^{-1}$ ) (Ivy, 2002). However, failure to consume CHO in the immediate post exercise phase of recovery leads to very low rates of glycogen restoration ( $1\text{-}2\text{mmol.kg ww.h}^{-1}$ ) until feeding occurs (Ivy *et al.*, 1998). If ingestion of CHO is delayed for 2h or more, this phase of very rapid CHO synthesis is missed. In the face of substantial glycogen depletion, 20- 24h of well- fed recovery are required for the restoration of muscle glycogen stores.

#### *1.10.2 Glycaemic index of carbohydrate:*

The rapid restoration of muscle glycogen stores is a critical issue for athletes who undertake prolonged training or competition sessions on the same or successive days. Since glycogen storage is influenced by insulin and a rapid supply of glucose substrate, different types of CHO and CHO-rich foods appear to have different effects on rates of muscle glycogen synthesis. Accordingly, the success of various post exercise feeding programs in promoting glycogen storage had been well investigated (Blom *et al.*, 1987; Costill *et al.*, 1981; Ivy *et al.*, 1988a; 1988b; Keizer *et al.*, 1986; Kiens *et al.*, 1990; Reed *et al.*, 1989; Roberts *et al.*, 1988). Conventional approaches to the study of post exercise glycogen storage have focused on intravenous glucose infusions or oral feedings of glucose, fructose, sucrose, or glucose polymers (Blom *et al.*, 1987; Ivy *et al.*, 1988a; 1988b; Reed *et al.*, 1989). Studies using feedings of simple sugars have found that glucose and sucrose feedings produce similar rates of muscle glycogen recovery after exercise, whereas the intake of fructose produces a lower rate of glycogen storage in the exercised muscles because it is absorbed more slowly from the intestine. These older studies investigating the effect of different types of CHO-rich foods on glycogen storage failed to find consistent results, most probably because they used the confusing system of dividing CHO foods into simple or complex CHO foods on the basis of their chemical composition (Brewer *et al.*, 1988; Costill *et al.*, 1981; Roberts *et al.*, 1988). It has been

represented that ingestion of simple CHO foods will elicit a large, rapid and short lived rise in blood glucose, whereas the response to complex CHO foods will be flatter and more sustained. However, this system does not truly differentiate the effects of different CHO- rich foods on glucose and insulin responses. Additionally, in practice, athletes need to consume food, rather than single nutrients, to meet a complex array of nutritional requirements as well as for social and practical reasons. There are limited research examining the effects of CHO meals. In practice, athletes are more likely to eat a combination of foods, especially if the recovery time is longer than a few hours.

As previously mentioned the GI was introduced in order to describe more accurately the blood glucose responses to CHO foods (Frail and Burke, 1994). Since glycogen storage is influenced by both insulin and a rapid supply of glucose substrate, research has been conducted into the effects of the GI of CHO supplements on the restoration of muscle glycogen during post-exercise recovery (Burke *et al.*, 1993; Joszi *et al.*, 1997; Parkin *et al.*, 1997; Stevenson *et al.*, 2005; Erith *et al.*, 2006). The ingestion of a high GI CHO is generally associated with large glycaemic and insulinaemic responses. It is therefore logical that CHO sources with a HGI would enhance post exercise glycogen resynthesis (Burke *et al.*, 2004) and these types of foods are generally recommended to athletes, during recovery from exercise. The cause of reduced glycogen storage with LGI foods is unclear, but may be in part due to the relatively poor digestibility of many of these foods. A diet or meals based on indigestible CHO foods may overestimate the real amount of CHO that is available for glycogen storage by the muscle.

Several studies have examined the effect of HGI CHO drinks consumed during recovery from exercise (Ivy, 1998; Wong and Williams, 2000; Jentjens *et al.*, 2001), however, few have examined the effects of CHO meals and research into the effects of differing GI of foods on recovery and performance is equivocal as inconsistent findings have been reported.

Kiens *et al.* (1990) first studied CHO foods and muscle glycogen storage based on actual rather than assumed glycaemic responses. In this study Kiens *et al.* (1990) compared the levels of muscle glycogen restoration during a 44h post- exercise recovery period. Individuals consumed either a CHO- rich diet based on foods with a high GI or a CHO- rich diet consisting of low GI

foods after exercise. They reported that a high- CHO diet based on foods with a high GI produced greater storage of glycogen during 6h of recovery than a diet based on low GI CHO- rich foods. However, there was no difference in muscle glycogen levels between diets at 20, 32, and 44h of recovery. Plasma insulin levels were greater with the high GI diet during the first 6h, but glucose and insulin concentrations were similar at all other time points. There is some confusion interpreting these results however, as the diets provided to the participants were not reported and the foods were also described interchangeably as high GI / simple and low GI / complex (Burke *et al.*, 1993). Following this, Burke *et al.* (1993) examined the effect of the GI of post- exercise CHO intake on the muscle glycogen storage during a 24h recovery period. For this, five cyclists performed a 2h ride at 75%  $\dot{V}O_{2\max}$  followed by four, 30s all out sprints on two occasions, in order to deplete their muscle glycogen. For 24h after each trial, the cyclists rested and consumed diets of high CHO meals including either foods with a high GI or foods with a low GI. The total amount of CHO intake over the 24h was  $10\text{g}\cdot\text{kgbw}^{-1}$ . Muscle biopsy data showed that the consumption of HGI CHO meals resulted in higher muscle glycogen resynthesis than following the consumption of an isocaloric LGI diet. However, the magnitude of increase ( $\sim 30\%$ ) in glycogen storage with the HGI CHO trial was far greater than the increase in 24h blood glucose and insulin concentrations following the HGI trial. In other words, while the outcome of the study was as predicted, it could not be explained in terms of alteration of glucose and insulin responses. It would be reasonable to assume that endurance capacity would be greater during subsequent exercise although, this was not investigated.

Additionally, Joszi *et al.* (1996) investigated the effect of ingesting the CHO supplements with the different GI on resynthesis of the muscle glycogen during a 24h recovery period after glycogen depleting exercise. For this, eight endurance trained male cyclists performed a glycogen depleting exercise and rested for 24h, followed by a 30 min cycling TT. During the 24h recovery period, cyclists consumed glucose, maltodextrin (glucose polymer), waxy starch (100% amylopectin), or resistant starch (100% amylose). Glucose, maltodextrin and waxy starch were included in the high GI CHO supplement whereas the resistant starch was related to a low GI value.

It was found that the storage of muscle glycogen after a 24h recovery period from glycogen depleting exercise was greater after the high GI CHO- supplement than after the low GI CHO- supplement. There were no differences in total work output during the subsequent time trial between treatments. The authors suggested that the lower muscle glycogen storage after the low GI CHO- supplement may be due to the poor digestibility of the high- amylose starch mixture. The indigestible CHO forms provide a poor substrate for muscle glycogen resynthesis, overestimating the CHO that is actually available from the consumed meal. This issue needs to be further studied in relation to real foods as in this study, the authors used solutions of glucose, maltodextrins, and a high amylopectin starch (all high GI).

Wee *et al.* (2005) examined whether the GI of CHO- rich pre- exercise breakfast affects muscle glycogen storage during a 3h postprandial period and muscle glycogen utilization during subsequent exercise. For this, seven male recreation runners were provided with isocaloric breakfasts, which were either high or low GI and provided  $2.5\text{g.kgbw}^{-1}$ . Following a 3h postprandial period, each participant was then required to run at  $70\% \dot{V}O_{2\text{max}}$  for 30min. It was found that when a low GI breakfast was consumed 3h before exercise, less CHO was stored as muscle glycogen than when a high GI breakfast was consumed (Wee *et al.*, 2005). This 15% increase in muscle glycogen concentration was reported at the end of a 3h postprandial period after the high GI breakfast. However, only a small non- significant increase in muscle glycogen was reported after the low GI breakfast. This was accounted for primarily by the low glycaemic and insulinaemic responses to the low GI meal, followed by the slow digestion and absorption of the ingested foods. The major finding of this study was that when different pre- exercise breakfasts were consumed, a LGI meal contributed less CHO to the muscle glycogen stores during a 3h postprandial period compared with a HGI meal. However, plasma FFA availability and fat oxidation were better maintained in the LGI trial, together with a sparing of muscle glycogen utilisation and lower muscle lactate accumulation during 30 min of submaximal running (Wee *et al.*, 2005).

According to these studies, HGI foods seem to be the desirable CHO supplementation during the post- exercise recovery period. It could be that the consumption of high GI foods elicits

an increased rate of muscle glycogen resynthesis compared to the consumption of low GI foods. A possible explanation for this difference is that the high GI foods elicit greater substrate availability for glycogen resynthesis.

In contrast to these above findings, Stevenson *et al.* (2005) examined the effects of high GI and low GI CHO meals consumed during a 24h recovery period after glycogen depleting exercise on endurance capacity and the metabolic responses during exercise the following day. For this, nine male recreational athletes performed a glycogen depleting protocol, following which, they were provided with either high or low GI recovery diets, each providing 8g. kgbw<sup>-1</sup>. The following day, the participants were then required to run to exhaustion at 70%  $\dot{V}O_{2\max}$ . Participants ran significantly longer on the low GI (108.9min) recovery diet than they did on the high GI (96.9min) recovery diet. Greater free fatty acid concentrations and fat oxidation in the low GI trial were attributed to the improvement in endurance capacity observed in this study. It may have been that the higher fat oxidation observed in the low GI trial may have spared muscle glycogen during exercise, subsequently improving endurance performance. This study did however, have the athletes perform the time to exhaustion protocol in the fasted state in the morning, which would have altered metabolism and fuel oxidation in addition to the GI of the recovery diets. This factor needs to be considered when interpreting these findings.

Results from a study conducted by Erith *et al.* (2006) contrasted with those previously observed by Stevenson *et al.* (2005). Erith *et al.* (2006) found that providing participants with both high and low GI recovery diets had no influence on performance during subsequent prolonged high-intensity intermittent shuttle running. Similarly, following a 22h recovery, in which the participants consumed 8g·kgbw<sup>-1</sup> of CHO, there was no difference in time to fatigue ( $25.3 \pm 4.0\text{min}$  vs.  $22.9 \pm 5.6\text{min}$ ), sprint performance ( $43 \pm 7\text{s}$  vs.  $39 \pm 10\text{s}$ ) or distance covered ( $3474 \pm 531\text{m}$  vs.  $3097 \pm 793\text{m}$ ) between the high and low GI recovery diets, respectively. It was suggested that when on the high GI recovery diet the participants began intermittent exercise using more CHO than fat because of its greater availability (Roepstorff *et al.*, 2005), whereas after the low GI recovery diet they used



more fat and less CHO. The authors concluded that the GI of the diet during the 22h recovery period did not affect sprint performance the following day.

Parkin (1997) examined whether delaying the ingestion of high GI food by 2h had any effect on muscle glycogen storage at 8 and 24h following prolonged, exhaustive exercise. Six endurance trained men performed a glycogen depleting protocol on two separate occasions. They were then provided with isocaloric high GI meals, each providing  $2.5\text{g}\cdot\text{kgbw}^{-1}$  either immediately after the trial and then at 2, 4, 8, and 22h post exercise; or 2 h post- exercise and at 4, 6, 8, and 22h post-exercise. No differences were observed in muscle glycogen storage or glucose- 6- phosphate concentration at any time in the two trials. The results of this study suggest that providing that sufficient CHO is ingested during the recovery period, delaying feeding of a high GI meal by 2h has no effect on the rate of muscle glycogen re-synthesis at 8 and 24h post- exercise.

There is clear evidence that adequate CHO intake is important for the restoration of muscle glycogen stores, and that other dietary strategies related to the timing of intake, type of CHO source or addition of other nutrients may either directly enhance the rate of glycogen recovery or improve the practical achievement of CHO intake targets.

Research has been conducted into the effects of the ingestion of foods of different GI before exercise (Thomas *et al.*, 1991; 1994; DeMarco *et al.*, 1999) and during 24h recovery period following glycogen depleting exercise (Burke *et al.*, 1993; Stevenson *et al.*, 2005). Little research however, has been conducted into the effects of the GI of meals on shorter recovery periods between multiple bouts of exercise.

### **1.11 Limitations of Previous Research**

As previously discussed, consuming LGI foods before exercise has been shown to result in improvements in exercise performance (DeMarco *et al.*, 1999; kirwan *et al.*, 1998; 2001; Stevenson *et al.*, 2005; Thomas *et al.*, 1991; Wu and Williams, 2006). However, despite the plethora of research supporting the use of LGI meals on exercise performance, an equal amount of controversy surrounds this area, with studies demonstrating no significant difference in exercise performance

following the consumption of high compared to low GI foods (Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Stannard *et al.*, 2000; Thomas *et al.*, 1994; Wee *et al.*, 1999).

There have been several reasons postulated for this lack of agreement such as small differences in study design, particularly timing of the meal, exercise intensity, meal composition, and the specific metabolic responses to meals. However, an important factor in the interpretation of these studies, as with many areas of sports nutrition research, lies in the issue of defining and measuring 'performance' (Burke, 2002).

#### *1.11.1 Exercise Performance*

The issues with defining and measuring performance have been postulated as some studies have failed to show any performance benefits arising from the consumption of a low GI pre- event meal, even when metabolism has been altered throughout the exercise (Sparks *et al.*, 1998; Stannard *et al.*, 2000; Thomas *et al.* 1994). In one of the first studies on the influence of HGI and LGI CHO foods on exercise capacity, the LGI food appeared to improve endurance capacity to a greater extent than the HGI food (Thomas *et al.* 1991). In contrast, Febbraio and Stewart (1996) observed no difference in exercise performance between and LGI and HGI trial. Although these results do not confirm the benefits of a LGI CHO diet as reported by Thomas *et al.* (1991), a direct comparison cannot be made because different performance criteria were used in the two studies. Thomas *et al.* (1991) assessed endurance capacity, whereas, Febbraio and Stewart (1996) assessed total work done in a fixed time (endurance performance). Furthermore, it should be noted that some studies that report a beneficial exercise outcome following the lowering of the GI of the pre- event meal (DeMarco *et al.*, 1999; Stevenson *et al.*, 2005; Thomas *et al.*, 1991), have measured time to exhaustion at a fixed work rate as their interpretation of performance. However, not all studies using this protocol have show performance changes resulting from differences in the pre- exercise meal. These protocols lack ecological validity in competitive sport where a successful performance is determined by being able to complete a set amount of work or set distance in the shortest possible time, and where the athlete is free to choose and vary their work rate (Burke and Deakin, 2001). It

has been observed that 'time to exhaustion' protocols have a high co-efficient of variation with respect to time (McClellan *et al.*, 1995). Therefore, one outcome of choosing a dependant variable with a low degree of reliability is that it increases the risk of achieving type II error (failure to detect a real change) suggesting that a possible explanation for the failure of studies to detect improvements in endurance performance, associated with improved metabolic characteristics during prolonged exercise, is that small changes are masked by the inherent variability of the exercise task (Burke and Deakin, 2001). Additionally, Sporer and McKenzie (2007) demonstrated that 20 km TT performance is highly reproducible in competitive cyclists and that the low CV variance observed between trials, is comparable to that expected during actual performance in elite athletes. Additionally, research by Palmer *et al.* (1996) found that the time taken for laboratory simulated 20 km and 40 km TT rides were highly reproducible (CV  $1.1 \pm 0.9\%$  and  $1.0 \pm 0.5\%$ , respectively). A significant correlation ( $r = 0.98$ ,  $p < 0.001$ ) was also observed between laboratory and road race times, although road race times were typically 8% slower. These data indicate that the Kingcycle ergometry system can be used as a reliable method of assessing short term endurance cycling performance.

Most of the subsequent studies on high and low GI CHO pre-exercise foods have used similar cycling performance trials (DeMarco *et al.*, 1999; Febbraio *et al.*, 2000; Goddpaster *et al.*, 1996; Paul *et al.*, 1996; Sparks *et al.*, 1998; Stannard *et al.*, 2000). In only one of these studies was there a significant difference in performance times following the ingestion of a LGI pre-exercise meal. Demarco *et al.* (1999) reported an improvement in exercise time after their participants had eaten a LGI meal 30min before exercise that required them to cycle for 2h at 70%  $\dot{V}O_{2\max}$  and then at 100%  $\dot{V}O_{2\max}$  to exhaustion. The times to exhaustion were 206, 130, and 120s for the LGI, HGI and water trials respectively. The most positive results have been reported when exercise capacity tests have been employed in cycling (Kirwan *et al.*, 1998) and some (Stevenson *et al.*, 2005; Wu *et al.*, 2006) but not all (Wee *et al.*, 1999) running studies. It is therefore recommended that future studies and research into this area employ a performance that closely resembles competition and the physical demands placed on the athlete by their sport.

### 1.11.2 Dietary Composition

As well as issues in the definition and measuring performance, discrepancies in results may be due to the dietary composition of the test meals. Although some, more recent studies (Stevenson *et al.*, 2005; Wu *et al.*, 2003) have employed tighter controls on the test meals, providing each participant with iso- caloric and iso- nutrient test meals prior to exercise, there have been some studies that have only matched the CHO content of the meal, ignoring any metabolic effects of other nutrients. Such examples include research by Febbraio and Stewart (1996) and Thomas *et al.* (1991; 1994) who prescribed LGI and HGI test meals, each containing  $1\text{g}\cdot\text{kgbw}^{-1}$  but were not matched for either fat or protein content, nor were they iso- caloric. These data, and the applicability of their subsequent findings do, therefore need to be interpreted with caution as it is not known how much of the enhanced or altered metabolic responses can be attributed to increased protein or energy contents of these test meals.

### 1.11.3 Timing of Ingestion

Another potential factor proposed for the variation in results was the timing of the meal; however, this does not appear to explain the differences adequately. Thomas *et al.* (1991) fed their participants 1h before exercise, and DeMarco *et al.* (1999) provided the meal 30min before exercise, but both saw a significant improvement in performance. Several researchers (Kirwan *et al.*, 1998, 2001; Febbraio and Stewart, 1996; Sparks *et al.*, 1998) fed their participants 45min prior to exercise and although Kirwan *et al.*, (1998, 2001) saw an improvement in performance, Febbraio and Stewart, (1996) and Sparks *et al.* (1998) reported no benefits. Furthermore, Wee *et al.* (1999) provided the meal 3h before exercise and found no advantage between HGI (113min) and LGI (111min) meals, whereas, Wu and Williams (2006) demonstrated an improvement in endurance capacity following the ingestion LGI foods 3h prior to exercise.

Similarly, many of the previous studies in this area have provided single foods in the hour before exercise (Febbraio and Stewart, 1996, 2000; Sparks *et al.*, 1998; Stannard *et al.*, 2000; Thomas *et al.*, 1991), or exercised in the fasted state (Stevenson *et al.*, 2005), which again lacks

ecological validity as it is unlikely to reflect normal dietary behaviour as athletes are recommended to consume a high- CHO meal 3-4h before exercise, which, for many recreational athletes is usually at lunchtime, 3-4h after consuming breakfast (Hargreaves *et al.*, 2000). These conflicting data, regarding time and quantity of ingestion appear to offer no clear explanation of the differences in performance as there is no consistency between these variables and performance outcome.

Alternatively, differences between studies may be explained by variations in the level of dietary and exercise control exerted before the experimental trials. The study by Kirwan *et al.* (2001) is the only one to date, in which the participants were kept in residence in a metabolic unit for the days preceding each trial. All meals were provided to the participants, exercise was supervised in the laboratory, and the meals and exercise were replicated before each trial. In contrast, in the studies referenced above, the participants were required to record and replicate diets and exercise training accurately before each experimental trial. If the participants deviated from the diet and/ or exercise training, it would have added variability to the experimental outcome and may have contributed to the different results. Therefore, of the GI of test meals on exercise performance requires further investigation under conditions of controlled diet and activity levels to achieve more reliable and valid outcomes.

### **1.12 Metabolic Events during Exercise:**

The metabolic common denominator in those studies that have shown a clear improvement in endurance capacity following ingestion of LGI CHO pre- exercise meals appears to be a combination of a greater rate of fat oxidation and more stable plasma glucose concentrations during exercise (Williams and Serratos, 2006). An increased fat oxidation is accompanied by a reduction in the rate of CHO degradation, suggesting a possible sparing of the limited glycogen store in skeletal muscles. Some support for this suggestion is provided in a previously described study (Wee *et al.*, 2005) who observed a 15% in muscle glycogen content after the HGI meal, however, only a small non- significant increase in muscle glycogen was reported after the LGI breakfast. This was

accounted for by the low glycaemic and insulinaemic responses to the LGI meal (secondary to the slow digestion and absorption of the ingested foods). During the 30min treadmill run, however, the amount of muscle glycogen used was significantly greater after the HGI than after the LGI meal. Again, the rate of fat oxidation was greater in the LGI trial and plasma glucose was more stable during exercise than when compared to the HGI trial and a sparing of muscle glycogen during subsequent exercise was observed in the LGI trial, most likely as a result of better maintained fat oxidation.

It is interesting that in the studies that have shown enhanced exercise performance, it is apparent that glucose levels were significantly elevated over control levels (DeMarco *et al.*, 1999; Thomas *et al.*, 1991), whereas, in the studies that reported no improvements, glucose levels are not different from control levels (Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Wee *et al.*, 1999). In Kirwan *et al.* (2001) study, elevated glucose levels during the MOD- GI trial were also found compared with control values. Additionally, Febbraio and Stewart (1996) found that differences in the pre- exercise glucose and insulin responses to the high and low GI meals, consumed 45min before steady- state cycling, disappeared shortly after the onset of exercise. They observed no differences in total CHO oxidation over 2h of exercise between the two CHO treatments, and similar muscle glycogen utilisation in both CHO trials and a water trial. Suggesting that any glycaemic differences at the onset of exercise are short- lived and of transient consequence to metabolism.

Thus, it appears that the improvement in exercise performance is dependant on whether the meal can maintain adequate euglycemia during exercise to sustain CHO oxidation. However, this does not preclude the possibility that when lipolysis is severely inhibited, the relative contribution of CHO oxidation to the total fuel supply in exercise may place too great a burden on available supplies, blood glucose levels may decrease, and no exercise performance advantage ensues.

Although some studies have provided clear evidence of improved exercise capacity coinciding with glycogen sparing (Tzinzas and Williams, 1998) there are older (Coyle *et al.*, 1986) as well as more recent studies (Claassen *et al.*, 2005) that have reported improvements in endurance

capacity that cannot be explained simply by differences in muscle glycogen at the point of fatigue. The additional exercise time may well be linked to the continued optimum rate of hepatic glucose output later in exercise, and so simply focusing only on differences in muscle glycogen may provide a limited understanding of the mechanisms underpinning these improvements in exercise duration.

### **1.13 Conclusion:**

The effects of food sources of CHO, containing simple sugars, complex carbohydrates, fiber, fat and PRO, on the glycaemic response to ingestion, subsequent energy delivery during exercise, and ultimately exercise performance has not yet been characterized clearly (Kirwan *et al.*, 2001). Therefore, the suggestion that pre- exercise ingestion of LGI food would be advantageous during exercise is still under investigation.

Thomas *et al.* (1991) were amongst the first to show that meals with a low GI response (typically GI <40) were associated with enhanced exercise performance compared with HGI (GI > 70) meals with similar results been recently corroborated by Kirwan *et al.* (2001) and Wu and Williams (2006) with these data suggesting that there may be an optimal combination of CHO, dietary fiber, viscosity, and GI that results in an enhanced exercise performance. However, support for these observations has not been consistent and subsequent studies have shown no beneficial effect of either HGI or LGI meals on time- trial performance or on time to exhaustion (Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Wee *et al.*, 1999).

There are several study limitations proposed for the lack of consistency throughout and these include the performance trial itself; is it representative of actual performance and does the intensity of the exercise challenge the glycogen stores sufficiently to observe any effects on performance? The composition of the test meals also requires better control and the diets should both be iso- caloric and iso- nutrient as well as being representative of actual athletic dietary practices with regards the timing and amount of ingestion. Therefore future research in this area needs to consider and address these issues.

The studies that have been conducted to examine the influence of the consumption of the GI of meals before exercise, have observed enhanced exercise performance when the LGI foods were consumed (Demarco *et al.*, 1999; Kirwan *et al.*, 1998; 2001; Thomas *et al.*, 1991). Of the studies that showed no improvement in subsequent exercise performance after the consumption of LGI meals, some still displayed similar metabolic and physiological consequences to those studies that observed improvements in performance (Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Stannard *et al.*, 2000; Thomas *et al.*, 1994; Wee *et al.*, 1999). These findings indicate that the LGI foods may have the potential benefit over HGI foods when considering the intake of CHO prior to exercises because of the promotion of sustained CHO availability during exercise. However, the significance of using pre- exercise GI foods for feedings in determining CHO availability during exercises and how this then translates into enhanced athletic performance remains elusive and requires further investigation.

#### **1.14 Study Aims:**

Based on the inconsistencies and limitations of the aforementioned research into the effects of the glycaemic index on exercise performance, the following three aims were elucidated:

##### **Aims:**

- 1) To determine the effects of consuming a pre- exercise low GI or high GI meal on metabolism and subsequent endurance performance.
- 2) To examine the effectiveness of high and low GI recovery diets, following prolonged exercise, on endurance performance.
- 3) Investigate the GI of recovery meals on metabolism and subsequent TT performance, following a glycogen depleting protocol and during a short term (3h) recovery period.



## **Novelty:**

- 1) Previous research carried out in this area is conflicting due to weaknesses in both the study designs and execution. This study will aim to strengthen past research in this area by improving the limitations of other studies to provide a more ecologically valid method that closely resembles the actual dietary and exercise practice of endurance athletes. Novel aspects to be covered include using an exercise protocol, which is representative of actual cycling time trial performance. Providing iso- nutrient test meals with a similar energy content at a time that resembles pre- match requirements and practice. Ensuring a normal dietary and exercise practice and environment is maintained at all times during the trial so to make the study as ecologically valid as possible.
- 2) Limited research has been conducted into post exercise nutrition, specifically the glycaemic index and endurance performance. Of the little research that has been carried out, endurance performance has not always been assessed and if so, has not always been representative of actual athletic performance. Therefore research into this area would be to add to and strengthen previous research, using a methodology that would be applicable to sports performance.
- 3) There is a lack of research into the effects of pre- exercise feedings on short term recovery between exercise bouts. One study- provided either LGI/ HGI 24h recovery meals both following and prior to participants performing the LIST with no differences in endurance capacity observed between the trials (Erith *et al.*, 2006). Therefore, there has been a call for more controlled, ecologically valid exercise tests to fill this gap in exercise protocols.

## **Chapter 2**

### **General Methodology**

#### **2.1 Introduction:**

This general methodology chapter describes the main equipment and materials that were used in the following studies. Any equipment or methodology that was used in a specific study is described within the methodology of that specific paper.

#### **2.2 Participants:**

All participants volunteered to participate in each study and were all competitive cyclists recruited from various sports clubs throughout the region. Participants were typically included in the research if they cycled five or more times a week, were free from any chronic disease, acute illness, or musculoskeletal injury, and were not taking any medication that might have affected their exercise response. On average, participants cycled ~120 miles per week and 40 k TT time ranged from 50-70 min.

The experimental procedures were described to each participant and they were each given the opportunity to ask questions. Each participant completed pre-exercise medical forms (Appendix 1) and informed consent forms (Appendix 2) before participating in any activity. Each study was granted ethical approval by the Department of Sport, Health and Exercise Science Ethics Committee at the University of Hull.

#### **2.3 Subject Preparation:**

Prior to participating in any experiment, each participant was required to sign a checklist (Appendix 3) that they had conformed to the requirements of the study and not engaged in any prohibited behaviour, which would have an influence on their performance. If conditions were not similar for each trial, for whatever reason, the participant was asked to return to the laboratory on another day when they had met the pre-exercise criteria described below. Participants were required to follow

the same diet and training schedule during the 2d prior to each experimental trial. Participants were asked to refrain from strenuous exercise and alcohol consumption in the 24h period before each experimental trial and caffeine intake was also prohibited 12h prior to each trial.

#### **2.4 Body Weight and Height Determination:**

Each participant had their height and weight measured on their initial visit to the laboratory and only their weight measured thereafter. Each participant's height was measured using a wall-mounted stadiometer (Holtain Ltd., Crymych, UK). The participant was required to stand with their feet together and their scapulae and heels making contact with the back of the wall and to look straight ahead. The participant took a deep breath and the sliding scale was lowered on to the top of their head. Height was recorded to the nearest 0.1cm.

Body weight was measured using balance beam scales (Seca Ltd., Hamburg) that were zero calibrated prior to each body weight measurement. Participants wore the same, minimal clothing and no footwear each time they were weighed. Body weight was measured to the nearest 0.1kg prior to each test.

#### **2.5 Ergometry:**

The performance measure used in each experiment involved a time trial (TT) of a specific distance, which was performed on either a Velotron CyclePro © (RacerMate Inc., Seattle, WA) or on an electro- magnetically braked cycle ergometer (Schoberer Rad Mebtechnik (SRM), Konigskamp, Germany), depending on the nature of the exercise involved. The Velotron cyclePro © ergometer has been previously found to have a low between trial coefficient of variation and a highly reproducible performance in competitive cyclists (Sporer and McKenzie, 2007). All testing took place on the same Velotron cyclePro© and SRM ergometer. A gradient of 2% was programmed into the Velotron so to add to effect of cycling on a road, which may explain the TT times.

## 2.6 Maximal Oxygen Consumption:

Prior to all experimental testing, participants undertook a graded exercise test to determine their  $\dot{V}O_{2\max}$ . All this testing took place on the SRM (pg 52) in the laboratory. Following a 5min warm-up at 100W, the participants then performed an incremental maximal exercise test to exhaustion. The cycling workloads began at 100W and increased 25W each minute until participants reached volitional exhaustion. The  $VO_{2\max}$  was deemed to have been elicited if two of the following criteria had been satisfied: a plateau in oxygen consumption, a respiratory exchange ratio (RER) >1.10, or a heart rate (HR) within 10 beats·min<sup>-1</sup> of age-predicted maximum heart rate (220 – age) (Midgley *et al.*, 2007).

## 2.7 Expired Air Analysis:

During each study, expired air was measured continuously breath-by-breath using an automated open-circuit gas analysis system (Quark b<sup>2</sup>, Cosmed Srl, Rome, Italy). The oxygen analyser is zirconia temperature controlled and has a response time of less than 120ms and an accuracy of 0.01%. The gas analysers were calibrated immediately prior to each test using ambient air (assumed to contain 20.94% oxygen (O<sub>2</sub>) and 0.03% carbon dioxide (CO<sub>2</sub>), and certified alpha standard gases containing 16.00% O<sub>2</sub> and 4.96% CO<sub>2</sub> (Cryoservice Ltd, Worcester, UK). The turbine flow meter used for the determination of minute ventilation has a resistance of <0.07 cmH<sub>2</sub>O L·S<sup>-1</sup> at a flow rate of 12L·s<sup>-1</sup>, an accuracy of ± 2%, and was calibrated with a 3-L syringe (Cosmed Srl, Rome, Italy) immediately before each test. Breath-by-breath data for each test were time-averaged over a specific time interval. All metabolic data were processed by the Cosmed data management software. From this data, substrate oxidation rates, energy expenditure and RER were obtained. Substrate oxidation rates and energy expenditure were calculated from  $\dot{V}O_2$  and  $\dot{V}CO_2$  values using stoichiometric equations (Frayn, 1983). The participants were required to wear the mask of the Cosmed only during each sampling point throughout the trials and not the whole time.

## **2.8 Heart Rate Monitoring:**

Heart rate was monitored continuously throughout all testing periods using a telemetric system. A heart rate transmitter belt (T41, Polar Electro, Kempele, Finland) was fitted around the participant's chest.

The transmitter belt electrodes were coated with electro-conductive gel (e.c.g. gel, Meditec, Italy) to enhance signal detection. The heart rate receiver is built into the Cosmed Quark b<sup>2</sup> metabolic cart and the heart rate data was processed by the Cosmed data management software.

## **2.9 Blood sampling:**

### *Cannulation:*

Before any blood was ever sampled, the sampling site was first cleaned using an alcohol wipe. Blood samples were collected using a butterfly cannula (BD Venflon™ IV Catheter, 20G, (pink), PTFE, ported, winged, 45mm, sterile, latex free) (BD Becton Dickinson, UK Ltd, Oxford, UK), which was inserted into an antecubital vein to allow for continuous blood sampling. The cannula was kept patent by flushing with saline immediately after it was inserted and after each blood collection.

### *Venous samples:*

Venous blood samples were taken aseptically from the antecubital vein and dispensed immediately into two different vacutainers. A specific amount was dispensed into a potassium EDTA Vacuette tube (Vacuette®, Greiner BIO-one, UK). The remainder was dispensed into a serum separator, clot-activator Vacuette tube (Vacuette®, Greiner BIO-one, UK).

The FFA was measured in duplicate (NEFA C, Wako Chemicals, kit number 999- 75406, Germany), and sample values were obtained by reference to an oleic acid standard supplied with the kit. They were then analysed according to manufacturer's instructions.

Triglyceride was measured in duplicate using an Infinity kit (kit number 7500- 023A) from Thermo and analysed according to the manufacturers' instructions.

Insulin values were determined by chemiluminescent immunoassay (Immulin 1000 Insulin Kit and Immulin 1000 analyser; Siemens DPC, USA).

Whole blood glucose and lactate were analysed using a lactate and glucose analyser (YSI 2700 Stat, Yellow Springs Instrument, Yellow Springs, USA). The coefficient of variation for glucose and lactate analysis in the laboratory and on this instrument is <3.8% for both analytes.

Osmolality was measured using a Micro osmometer model 3320 (Advanced Instruments Inc. Massachusetts, USA).

### **2.10 Environmental Testing Conditions:**

All testing took place within the Applied Physiology Laboratory of the Sport, Health and Exercise Science Department, University of Hull. All repeat trials were performed at the same time of day and under similar experimental and environmental conditions. The temperature, relative humidity and barometric pressure during each experiment of the laboratory were maintained at approximately 20- 24°C, 25- 50% and 744- 772mmHg, respectively.

### **2.11 Statistical Analysis:**

All statistical analyses were conducted using SPSS<sup>®</sup> for Windows software version 14.0 (SPSS Inc., Chicago, IL, USA). All statistical assumptions were checked and deemed plausible. Sample data are presented as the mean  $\pm$  standard deviation. All statistical analysis has been described separately within each study.

## Chapter 3

# Effect of the glycaemic index of a pre-exercise meal on metabolism and cycling time trial performance

### 3.1 Introduction

The importance of adequate dietary CHO for optimal endurance performance has long been recognised (Coggan and Coyle, 1991), but the type and amount of CHO might also impact on physical performance. Many studies have investigated the effects of pre-exercise meals (consumed immediately prior to or up to 3h before exercise) that differ in glycaemic index (GI) on physical performance. Researchers have demonstrated that low GI meals significantly improve physical performance (Thomas *et al.*, 1991; DeMarco *et al.*, 1999; Kirwan *et al.*, 1998; 2001; Stevenson *et al.*, 2005; Wu and Williams, 2006). These improvements are typically attributed to attenuated glycaemic and insulinaemic responses, increased free fatty acid concentrations and fat oxidation rates, and an associated decrease in muscle glycogenolytic rate. Some studies observed no differences in physical performance following the ingestion of either high or low GI pre-exercise meals (Sparks *et al.*, 1998; Febbraio and Stewart, 1996; Stannard *et al.*, 2000; Thomas *et al.*, 1994), which has been attributed to differences in study design, (Kirwan *et al.*, 2001).

A major limitation to the interpretation of previous research is that one or more aspects of the research design lacked some degree of internal or ecological validity. Arguably the most important aspect that limits the interpretation of previous research is that performance was assessed either by the total work completed in a fixed time (Sparks *et al.*, 1998; Febbraio and Stewart, 1996), time to exhaustion at a fixed work rate (Thomas *et al.*, 1991; Kirwan *et al.*, 1998, 2001; Stevenson *et al.*, 2005; Wu and Williams, 2006), or a combination of the two methods (DeMarco *et al.*, 1999). Since performance is almost invariably measured as time to complete a fixed task the ecological validity of the performance measures used in previous studies is questionable. The lack of validity is highlighted by the large improvements in performance of 11% to 59% for trained athletes, as

such improvements, due to dietary intervention appears unrealistic. Furthermore, some studies used test meals that were not iso-caloric or iso-nutrient, with only the CHO content being matched for the high and low GI meals (Burke *et al.*, 1998; Thomas *et al.*, 1994). These studies are difficult to interpret as differences in the energy or macronutrient content of the high and low GI meals may affect the physiological and performance responses to these meals.

The purpose of the present study was to investigate the influence of the GI of a meal, on physical performance using experimental methods that address two of the major limitations of previous studies. This study used a measure of performance, a time trial, with high ecological validity (Sporer and McKenzie, 2007). Furthermore, we used meals that differed widely in their GI, but were closely matched in terms of energy, fat and protein content. It was hypothesized that due to the reduced glycaemic and insulinaemic responses typically associated with low GI meals, there would be a significant improvement in time trial performance associated with the consumption of a low GI versus high CHO meal.

## **3.2 Methods**

### **3.2.1 Participants**

Ten well- trained male cyclists participated in the study. Their mean ( $\pm$  SD) age, height, weight, and  $\dot{V}O_{2\max}$  were  $28 \pm 6$ y,  $182.1 \pm 7.1$ cm,  $76.4 \pm 9.9$ kg, and  $58.2 \pm 10.1$ mL.kg<sup>-1</sup>.min<sup>-1</sup>, respectively.

### **3.2.2 Experimental Design**

Following a familiarisation trial, each participant undertook two 40km time trials (TT) on a Velotron cyclePro © (pg 52) separated by at least seven days. Two different CHO test meals providing 1g.kgbw<sup>-1</sup> of CHO were ingested 45min prior to the TT. This study was carried out in a randomised, single- blind, cross-over fashion. The pre-exercise test meals were consumed while the participants were seated in the laboratory.



### 3.2.3 Maximal Oxygen Consumption ( $\dot{V}O_{2\max}$ )

Prior to all experimental testing, participants undertook a graded exercise test to determine their maximal oxygen uptake ( $\dot{V}O_{2\max}$ ). All testing took place on the same SRM, at the same time of day in the laboratory. Expired air was measured breath- by- breath using an automated open- circuit gas analysis system. The equipment and procedure for this maximal test has previously been described in chapter 2 (pg 53).

### 3.2.4 Experimental Protocol

Each participant reported to the laboratory 6h post- prandial and after sitting for 5min, a basal venous blood sample and a 5min resting expired air sample was collected via standard open circuit spirometry (pg 53). They were then provided with a standardised meal that provided  $1\text{g}\cdot\text{kgbm}^{-1}$  of CHO which was composed of either high or low GI carbohydrates. The participants consumed the meal within 10min and then rested for 45min before they commenced the exercise protocol. A 15min post-prandial venous blood sample was then collected and a heart rate (HR) monitor (pg 54) was attached to the participant. A final pre exercise venous blood sample was obtained 10min before the onset of exercise.

Following a similar, pre-competition warm up, consisting of approximately 5min cycling at 50W, the participants were required to complete the 40km TT in as short a time as possible. Throughout the TT, 5min expired air samples were collected during the last 5min of every 20min time period and venous blood samples were collected. The final expired air sample was collected in the last minute of cycling and the last venous blood sample was taken immediately following termination of the trial while participants were still seated on the ergometer. Heart rate and ratings of perceived exertion (RPE), using Borg 6- 20 point scale (Borg, 1982), were recorded every 15min. Participants were not aware of their performance times and HR but they were given a verbal reminder every 15min to continue until they reached the set distance or their limit of exercise tolerance.

3.2.5 Test Meals

The high and low GI meals were consumed in the laboratory 45min before the onset of exercise, and participants were unaware of the type of meal they had consumed until after completion of the study. The test meals were similar in macronutrient content (Table 3.1). The high GI meal consisted of Cornflakes (Kelloggs Co., Manchester, UK) and semi-skimmed milk (GI: 72) whereas the low GI meal consisted of Branflakes (Kelloggs Co., Manchester, UK) and semi-skimmed milk (GI: 30). The GI values of each test food were taken from Henry *et al.* (2005) and the GI of the total meal was calculated from the weighted means of the GI values for the component foods (Wolever and Jenkins, 1986). To ensure each participant consumed a standardised volume of fluid (650ml), they consumed a specific amount of water and milk with each meal.

**Table 3.1**  
Characteristics of a test meal for a 70kg participant.

|              |                  | High- GI breakfast                                    | Low-GI breakfast                                      |
|--------------|------------------|---|---|
| Description  |                  | 68g Cornflakes, 282ml semi-skimmed milk, 368ml water. | 81g Branflakes, 337ml semi-skimmed milk, 313ml water. |
| Meal content | Energy (kcal)    | 386   | 422   |
|              | Carbohydrate (g) | 70  | 70  |
|              | Fat (g)          | 6   | 8   |
|              | Protein (g)      | 16  | 19  |
|              | Fibre (g)        | 2   | 12  |
|              | Glycaemic index  | 72*   | 30*   |

Macronutrient breakdown of high GI and low GI test meals provided to each participant were calculated from manufacturer’s information. \*Calculated by the method of Wolever and Jenkins (1986). Glycaemic index values from Henry *et al.* 2005).

**Sample collection and analysis:**

3.2.6 Gas analysis:

Expired air was measured breath- by- breath using an automated open- circuit gas analysis system. The equipment employed in gas analysis has previously been described in chapter 2 (pg 53).

### 3.2.7 Blood analysis

Blood samples were collected using a butterfly cannula inserted into an antecubital vein to allow for continuous blood sampling. During the trial, 12ml blood samples were obtained at each sampling point, and immediately dispensed into two potassium EDTA Vacuette tubes. Then, 6ml was immediately analysed for whole blood glucose and lactate as previously described (pg 54). The remainder was immediately centrifuged at 13000 x g for 10min and analysed for plasma osmolality. The aliquoted plasma was then stored at -85°C and later analysed for insulin, FFA and triglycerides. These equipment and procedures involved in the analysis of these hormones and substrates have been previously described in chapter 2 (pg 54).

### 3.2.8 Data Analysis

All statistical analyses were performed using SPSS® for Windows software (v 16.0; SPSS Inc., Chicago, IL). A paired samples t test was used to assess any differences in baseline data on all the variables prior to any further analysis taking place. A repeated measures analysis of variance (ANOVA) on two factors (trial and time) was used to determine metabolic and performance differences between trials. In the event of a significant F ratio, Sidak-adjusted post hoc tests were used to locate significant paired differences. Differences were considered statistically significant at the  $p < 0.05$  level. All data are presented as mean  $\pm$  standard deviation.

## 3.3 Results

### *TT performance time*

The average TT time in the low GI trial ( $93 \pm 8\text{min}$ ) was significantly shorter than the high GI trial ( $96 \pm 7\text{min}$ ) ( $t = -3.3$ ;  $p = 0.009$ ; 95% confidence interval for the mean difference  $-4.9$  to  $-0.9\text{min}$ ),  $\Omega = 0.33$ .

### Heart rate and RPE

There was a significant main effect for time ( $F = 465.6$ ,  $p < 0.001$ ) but not trial ( $F = 0.1$ ,  $p = 0.81$ ) and no significant trial-by-time interaction for heart rate ( $F = 1.6$ ,  $p = 0.17$ ) (Table 3.2). The post-hoc tests for heart rate revealed that time points one (baseline) and time point two (45 min pp) were significantly different to all other time points ( $p = < 0.001$ ). There was a significant main effect for both time ( $F = 200.2$ ,  $p < 0.001$ ) and trial ( $F = 5.8$ ,  $p = 0.039$ ) for RPE, where RPE was lower in the low GI trial and increased over time to a maximum value of 20 and 18 for the high GI and low GI trials, respectively. There was no significant trial-by-time interaction for RPE ( $F = 1.6$ ,  $p = 0.17$ ). The post-hoc tests for RPE revealed that time points one (Baseline) and time point two (45 min pp) were significantly different to all other time points ( $p = < 0.001$ ). Time point three (20 min) was also significantly different from time point five (60 min) ( $p = 0.009$ ).

**Table 3.2:** Oxygen uptake ( $\text{VO}_2$ ), heart rate, and the respiratory exchange ratio (RER) during the high glycaemic index (HGI) and low glycaemic index (LGI) CHO trials. Data are presented as means  $\pm$  SD of the mean. pp = post- prandial.

| Variable               | Trial | Baseline           | 45pp               | 20                | 40                | 60                | End               |
|------------------------|-------|--------------------|--------------------|-------------------|-------------------|-------------------|-------------------|
| $\text{VO}_2$          | HGI   | $0.33 \pm 0.06$    | $0.41 \pm 0.11$    | $3.02 \pm 0.49$   | $2.83 \pm 0.50$   | $3.13 \pm 0.44$   | $3.11 \pm 0.43$   |
| (L.min <sup>-1</sup> ) | LGI   | $0.31 \pm 0.05$    | $0.39 \pm 0.12$    | $3.18 \pm 0.30$   | $2.99 \pm 0.35$   | $3.0 \pm 0.22$    | $3.37 \pm 0.19$   |
|                        | HGI   | $0.86 \pm 0.05$    | $0.87 \pm 0.04^*$  | $0.94 \pm 0.04^*$ | $0.92 \pm 0.02$   | $0.89 \pm 0.03^*$ | $0.94 \pm 0.01$   |
| RER                    | LGI   | $0.9 \pm 0.09$     | $0.92 \pm 0.04^*$  | $0.98 \pm 0.03^*$ | $0.93 \pm 0.02$   | $0.91 \pm 0.02^*$ | $0.97 \pm 0.04$   |
| HR                     | HGI   | $62.2 \pm 12.01^*$ | $63.6 \pm 12.22^*$ | $164.1 \pm 15.07$ | $164.4 \pm 15.71$ | $163.2 \pm 13.55$ | $170.5 \pm 16.32$ |
| (b.min <sup>-1</sup> ) | LGI   | $60.1 \pm 12.67^*$ | $63.9 \pm 10.59^*$ | $166.9 \pm 7.87$  | $166.9 \pm 7.87$  | $170.1 \pm 7.64$  | $163 \pm 22.66$   |

\* Significantly different ( $P < 0.05$ ) at 45 min pp to 20 and 60 min for RER. RER was also significantly different from 20 min to 40 min and from 60 min to the end within trials.

\* HR was significantly different ( $P < 0.05$ ) at baseline and 45 min to all other time points within trial.

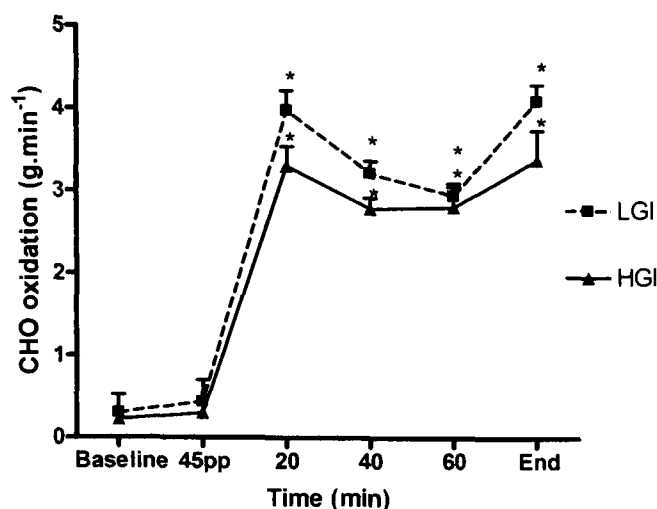
### *VO<sub>2</sub> and Respiratory Exchange Ratio (RER) (Table 3.2)*

There was no significant difference in  $\dot{V}O_2$  between the high GI ( $71 \pm 9\% \dot{V}O_{2\max}$ ) and low GI trial ( $72 \pm 10\% \dot{V}O_{2\max}$ ) ( $t = 4.0$ ;  $p = 0.88$ ). There was a significant main effect for both time ( $F = 11.2$ ,  $p = 0.001$ ) and trial ( $F = 24.2$ ,  $p < 0.001$ ) for RER. No significant trial-by-time interaction was observed for RER ( $F = 1.5$ ,  $p = 0.22$ ). The post- hoc tests for RER revealed that the time point two (45 min) was significantly different to time points: 3 ( $p = 0.16$ ) and 5 ( $p = 0.008$ ). Time point 3 (20 min) is significantly different from time point 4 ( $p = 0.003$ ) and time point 5 ( $p = 0.014$ ). Time point 5 was also significantly different from time point 6 ( $p = 0.013$ ).

### *Substrate Utilisation*

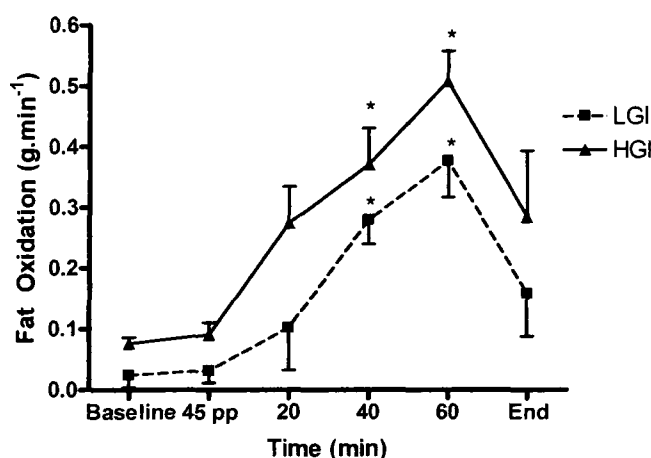
There was a significant main effect for both time ( $F = 75.1$ ,  $p < 0.001$ ) and trial ( $F = 15.7$ ,  $p = 0.003$ ) (Figure 3.1), but no significant trial-by-time interaction for CHO oxidation rates ( $F = 1.6$ ,  $p = 0.2$ ). The post- hoc tests for CHO oxidation revealed that the time point two (45 min pp) was significantly different to time points: 3 ( $p = < 0.001$ ), 4 ( $p = < 0.001$ ), 5 ( $p = < 0.001$ ), and 6 ( $p = < 0.001$ ). Time point three was also significantly different to time point 4 ( $p = 0.011$ ). A significant main effect for both time ( $F = 11.2$ ,  $p < 0.001$ ) and trial ( $F = 20.1$ ,  $p = 0.002$ ) (Figure 3.2) was observed for fat oxidation rates, but no significant trial-by-time interaction ( $F = 0.4$ ,  $p = 0.83$ ). The post- hoc tests for fat oxidation revealed that the time point two (45 min pp) was significantly different to time points: 4 ( $p = < 0.001$ ) and 5 ( $p = < 0.001$ ). Time point 5 (60 min) was also significantly different from time point 6 ( $p = 0.038$ ).

**Figure 3.1** Carbohydrate oxidation throughout the trial. Data are presented as means  $\pm$  SEM. pp = post-prandial.



\* Significantly different from 45pp for across all time points ( $p < 0.05$ )

**Figure 3.2** Fat oxidation throughout the trial. Data are presented as means  $\pm$  SEM. pp = post-prandial.



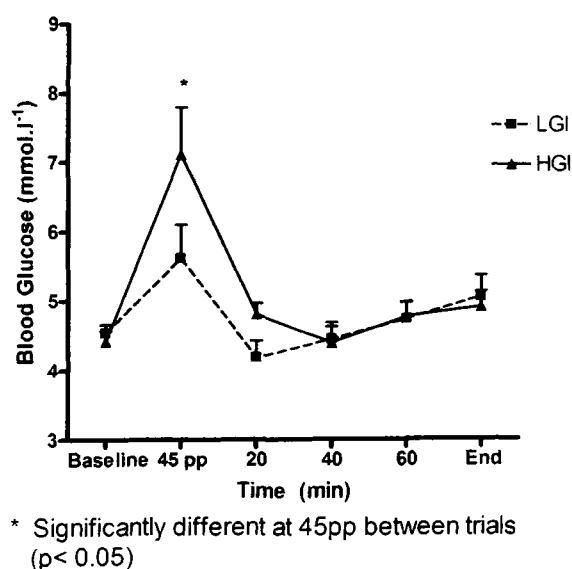
\* Significantly different from 45pp ( $p < 0.05$ )

#### *Whole Blood Glucose, Free Fatty Acids (FFA), Triglycerides (TGA)*

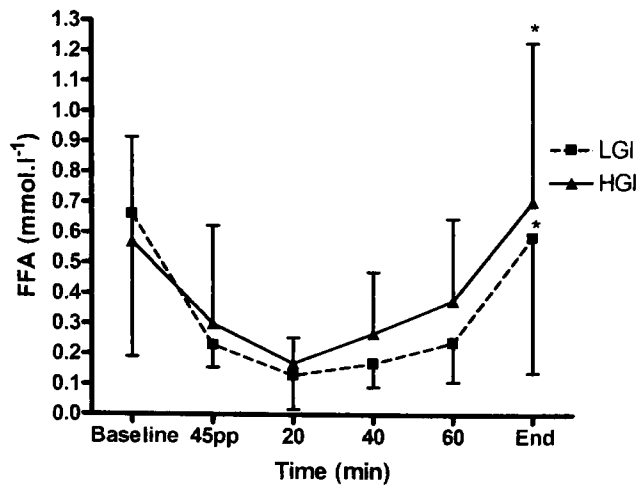
There were no significant differences in baseline values between trials for whole blood glucose ( $t = 0.6$ ,  $p = 0.58$ ), FFA ( $t = 0.2$ ,  $p = 0.85$ ), or TGA ( $t = 0.3$ ,  $p = 0.74$ ). Following the consumption of the high GI meal, the whole blood glucose concentration rose significantly from  $4.42 \pm 0.49$  to  $7.11 \pm 2.16 \text{ mmol} \cdot \text{l}^{-1}$  for the high GI trial compared to  $4.54 \pm 0.37$  to  $5.61 \pm 1.55 \text{ mmol} \cdot \text{l}^{-1}$  for the low GI trial. During the TT the mean blood glucose concentrations were maintained between 4 and

5mmol·l<sup>-1</sup> for both the low GI and high GI trials. However, during both the low GI and high GI trials three participants experienced a drop in whole blood glucose levels below 3.5mmol·l<sup>-1</sup>. One athlete became hypoglycaemic 20min into the LGI trial, one athlete at 40min into both trials, and one athlete during the last 3min of the LGI trial. No symptoms of hypoglycaemia were reported. There was a significant main effect for time ( $F = 7.7$ ,  $p = 0.008$ ) but not trial for whole blood glucose ( $F = 2.2$ ,  $p = 0.17$ ) (Figure 3.3). There was a significant trial-by-time interaction effect for whole blood glucose concentration ( $F = 6.9$ ,  $p < 0.001$ ), where the post- prandial glucose concentration was significantly different between trials ( $p = 0.018$ ). There was a significant main effect for time ( $F = 9.0$ ,  $p < 0.001$ ) but not trial ( $F = 2.8$ ,  $p = 0.16$ ) for FFA (Figure 3.4), and no significant trial-by-time interaction ( $F = 0.2$ ,  $p = 0.91$ ). The post- hoc tests for FFA concentration revealed that the time point two (45 min pp) was significantly different to time point 5 ( $p = 0.011$ ) and time point three (20 min) was significantly different t time point 5 ( $p = 0.033$ ). There was no significant main effect for time ( $F = 1.2$ ,  $p = 0.35$ ) or trial ( $F = 1.9$ ,  $p = 0.23$ ) and no significant trial-by-time interaction for TGA ( $F = 1.1$ ,  $p = 0.40$ ).

**Figure 3.3** Whole blood glucose concentration throughout the trial. Data are presented as means  $\pm$  SEM. pp = post- prandial.



**Figure 3.4** Free fatty acid concentration throughout the trial. Data are presented as means  $\pm$  SEM. pp = post- prandial.



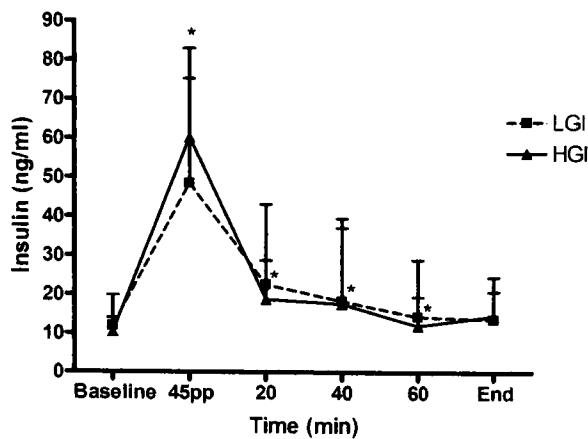
\* Significantly different at the End from 20 and 40 mins  
p < 0.05

### *Plasma insulin*

There was no significant difference in baseline values between trials for insulin ( $t = 0.34$ ,  $p = 0.74$ ). Following the ingestion of the HGI meal the insulin concentration was significantly higher (60.3ng/ml) than after the low GI meal (48.4ng/ml) ( $p = 0.008$ ). There was a significant main effect for both time ( $F = 33.7$ ,  $p < 0.001$ ) and trial ( $F = 17.4$ ,  $p = 0.014$ ) (Figure 3.5) and a significant trial-by-time interaction ( $F = 11.3$ ,  $p < 0.001$ ) for insulin. The post- hoc tests for insulin revealed that the time point one (45 min pp) was significantly different to time points: 2 ( $p = 0.014$ ), 3 ( $p = 0.022$ ) and 4 ( $p = 0.008$ ). The post-prandial insulin concentration was significantly different between trials ( $p = 0.008$ ), with the high GI meal being significantly greater (60.3ng.ml) than the low GI meal (48.4ng.ml).



**Figure 3.5** Insulin concentrations throughout the trial. Data are presented as means  $\pm$  SEM. pp = post- prandial.



\* Significantly different between high and low GI at 45pp. Significantly different at 20, 40, 60min for the high GI trial from 45pp ( $p < 0.05$ ) (this did not quite reach statistical significance for the low GI trial).

### *Whole Blood Lactate*

No significant difference was observed between pre-exercise whole blood lactate concentrations for the high GI ( $1.14 \pm 0.48 \text{ mmol} \cdot \text{l}^{-1}$ ) and low GI trials ( $1.07 \pm 0.52 \text{ mmol} \cdot \text{l}^{-1}$ ) ( $t = 0.4$ ,  $p = 0.73$ ). There was a significant main effect for time ( $F = 26.4$ ,  $p < 0.001$ ) but not trial ( $F = 0.8$ ,  $p = 0.39$ ) and no significant trial-by-time interaction for whole blood lactate concentration ( $F = 1.5$ ,  $p = 0.21$ ). The post- hoc tests for lactate concentration revealed that the time point two (45 min pp) was significantly different to time points: 3 ( $p = < 0.001$ ), 4 ( $p = < 0.001$ ), 5 ( $p = < 0.001$ ) and 6 ( $p = < 0.001$ ). Time point 3 (20 min) was significantly different to time point 4 ( $p = 0.016$ ) and 5 ( $p = 0.012$ ). Time point 5 (60 min) was significantly different to time point 6 ( $p = < 0.001$ ).

### *Osmolality*

Pre- and post -TT osmolality values for the LGI were  $294 \pm 3$  and  $298 \pm 4$ , respectively. Pre- and post -TT osmolality values for the HGI were  $297 \pm 10$  and  $296 \pm 5$ , respectively.

### 3.4 Discussion

Data from the present study show that, ingestion of a low GI CHO meal 45min prior to the onset of exercise can improve endurance performance during a 40-km TT by an average of 3min (3.2%), when compared to the consumption of a high GI CHO meal. At the 95% confidence level, the plausible range for the true population mean reduction in performance ranged from 0.9 to 4.9min. We would suggest that even a 0.9min reduction in performance time is of practical significance to well-trained cyclists. Although these data corroborate previous research in this area that also observed an improved performance with low GI foods (Thomas *et al.*, 1991; DeMarco *et al.*, 1999; Kirwan *et al.*, 1998, 2001; Stevenson *et al.*, 2005), the 3.2% improvement in performance appears somewhat lower than the 7.9% to 59% range of improvement in performance previously observed. This difference may be an indication of the reduced ecological validity of the protocols employed in these studies and how, possibly, they overestimate the beneficial effect that low GI meals are actually having on performance. Hopkins *et al.*, (2001) explains that a small change in an individual's power output results in a large change in the time to exhaustion. Nonetheless, from these studies, it would appear that low and/or moderate GI meals confer a performance advantage over a high GI meal or water. However, support for this observation has not been consistent and subsequent studies have shown no beneficial effect of either high GI or low GI meals on exercise performance (Sparks *et al.*, 1998; Febbraio and Stewart, 1996 Wee *et al.*, 1999).

Although discrepancies between these results may be explained by the differences in experimental methodologies, they may also be explained, in part, by inconsistent metabolic responses elicited by the varying pre-exercise meals. The metabolic and performance benefits resulting from pre exercise nutritional supplementation in the hour before exercise are unclear as nutritional supplementation with CHO before exercise has been shown to have an unpredictable effect on exercise performance and muscle fatigue (Wee *et al.*, 1999; Foster *et al.*, 1979; Hargreaves *et al.*, 1985; Hargreaves *et al.*, 1987; Neufer *et al.*, 1987).

It has been suggested that a hyperinsulinaemic state at the start of the exercise, is associated with a sustained suppression of FFA and increased usage of CHO as fuel early in exercise (Kirwan

*et al.*, 1998; 2001; Foster *et al.*, 1979). This may stimulate greater rates of glycogenolysis and early depletion of stored glycogen, leading to hypoglycaemia, which, as previously described, is believed to be a cause of fatigue during prolonged exercise (Kirwan *et al.*, 1998; Coyle *et al.*, 1986; Hermansen *et al.*, 1967). In the present study we expected that the insulin response elicited by the low glycaemic meal would be such that the antilipolytic effects of insulin on fat metabolism would be minimized and endogenous glucose would be preserved. We anticipated that the substrates oxidized during the early phase of exercise would be derived from the test meal and/or greater fat utilization, rather than glycogenolysis. However, the metabolic responses observed in this study are not consistent with those previously observed and make the interpretation of the performance results quite difficult. Unlike the typical inhibition of FFA and increased CHO oxidation observed previously with the consumption of high GI pre- exercise meals, CHO oxidation and RER was actually greater at this time and circulating levels of FFA were suppressed in the low GI trial. Both RER and CHO oxidation data indicate that CHO oxidation was greater throughout the trial following the ingestion of the low GI test meal. Moreover, circulating levels of FFA were suppressed at this time. Since FFA utilization during exercise is a function of circulating FFA concentration, this suggests a reduced opportunity for fat oxidation and a subsequent increased reliance on CHO sources. The enhanced performance for the low GI trial could be due to the greater CHO oxidation observed in the low GI trial, which suggests that the meal contributed additional CHO for oxidation and subsequently spared limited muscle and liver glycogen stores as previously suggested (Kirwan *et al.*, 2001; Jeukendrup *et al.*, 1997; Neuffer *et al.*, 1987). This additional CHO may have also provided a greater contribution of exogenous glucose to fuel use during the exercise bout (Neuffer *et al.*, 1987; Wright *et al.*, 1991). Although this observation could help explain the findings in this study, they are not consistent with those previously observed. These differences in results could be due to the sampling time points, being 20 min apart, which could have led important data going unnoticed. Despite that, the increased CHO oxidation observed in the low GI trial, suggesting that the meal contributed additional CHO to be used is plausible and could help to explain the findings observed.

### **3.5 Conclusion**

The ingestion of a low GI meal, containing  $1\text{g}\cdot\text{kgbw}^{-1}$  of CHO, 45min before performing a 40km TT protocol, significantly improved performance. This may suggest that the ingestion of the low GI food led to an increase in the availability of CHO and greater CHO oxidation throughout the exercise period. When these factors are combined with the possible delayed emptying of the ingested glucose from the gut, there may have been a glucose- sparing effect that facilitated sustained energy production towards the end of exercise. These data support the ingestion of a small meal with a low GI content before prolonged exercise.

## Chapter 4

### **The effects of low and high glycaemic index 24-h recovery diets on cycling time trial performance.**

#### **4.1 Introduction**

The ability to perform prolonged, exhaustive exercise is closely related to muscle glycogen stores (Bergstrom *et al.*, 1967; Coyle *et al.*, 1986). Athletes typically train or compete on consecutive days and therefore the rapid restoration of muscle glycogen stores during the recovery period is essential. The ingestion of a high quantity of CHO after exercise has repeatedly been shown to increase muscle glycogen repletion (Tsintzas *et al.*, 2003; Blom *et al.*, 1987; Ivy, 1998). Hence the consumption of high CHO foods or drinks after exercise is now common practice.

Different types of CHO foods appear to have different effects on rates of muscle glycogen synthesis depending on the insulin and glucose response to the CHO load (Doyle, Sherman and Strauss, 1993). Carbohydrate foods can be defined according to their postprandial glycaemic responses (Jenkins *et al.*, 1981) as either high GI or low GI. The ingestion of HGI foods is generally associated with a rapid increase in blood glucose and insulin concentrations, whereas the ingestion of LGI foods results in a flatter, more sustained glycaemic response (Burke, Collier, and Hargreaves, 1993). Since the rapidity of glycogen storage is influenced by both insulin and a rapid supply of glucose substrate, it is logical that CHO sources with a high GI would enhance post exercise glycogen resynthesis (Burke *et al.*, 2004).

Previous studies investigating the effect of different types of CHO rich foods on glycogen storage have failed to find consistent results, possibly because research has taken a simplistic approach to CHO nutrition, dividing foods into “simple” or “complex” CHO foods on the basis of their chemical composition (Brewer *et al.*, 1988; Costill *et al.*, 1981; Roberts *et al.*, 1988). However, this system does not truly differentiate the effects of different CHO- rich foods on glucose and insulin responses. In practice, however, athletes need to consume food, rather than single nutrients, to meet their recommended nutritional requirements as well as for practical

reasons. Therefore, using meals, Burke *et al.* (1993) demonstrated that consuming HGI CHO meals during a 24h recovery period from prolonged heavy exercise resulted in higher muscle glycogen resynthesis than following the consumption of an isocaloric LGI diet. However, exercise performance was not assessed in this study. Therefore, more recently, Stevenson *et al.* (2005a) conducted a study to examine the influences of high and low GI recovery diets on endurance running capacity after a recovery of 24h. Their participants ran longer on the low GI recovery diet than they did on the high GI recovery diet. A greater free fatty acid concentration and fat oxidation in the low GI trial was attributed to the improvement in endurance capacity observed in this study. A limitation of Stevenson's *et al.* (2005a) study however, is that they employed a time to exhaustion protocol, which has been associated with a high coefficient of variation with respect to time (McClellan *et al.*, 1995) and a lack of ecological validity.

However, results from a similar study conducted by Erith *et al.* (2006) contrast with those previously observed by Stevenson *et al.* (2005a). Erith *et al.* (2006) provided their participants with either high GI or low GI recovery diets over a 22h period following a high intensity shuttle- running protocol. No significant differences in glucose or free fatty acid concentration were observed between the diets and no improvement in time to fatigue, sprint performance or distance covered were observed between the two diets.

Previous research has produced inconsistent results examining post exercise performance and diet therefore, the purpose of the present study, was to examine the effect of high recovery CHO meals with different GI values on endurance performance, more specifically time trial cycling performance.

## **4.2 Methods**

### **4.2.1 Participants**

Ten well trained, but not elite male cyclists volunteered to participate in the study. Their mean ( $\pm$  SD) age, height, body weight, and  $\dot{V}O_{2\max}$  were  $33.6 \pm 7.4$ y,  $175.3 \pm 7.6$ cm,  $74.5 \pm 8.2$ kg, and  $60.5 \pm 6.0$ mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$  respectively.

#### **4.2.2 Experimental design**

Following a familiarisation trial, each participant performed two 40km time trials (TT) separated by at least seven days on a Velotron cyclePro © ergometer (pg 52). On day one of the experimental trial participants completed a glycogen depleting protocol. Following this protocol, one of two different CHO recovery diets providing  $8\text{g.kgbw}^{-1}$  of CHO were ingested over the next 24h. The participants returned to the laboratory on day 2 to perform a 40km TT on the Velotron cyclePro © ergometer. This study was carried out in a randomised, single- blind, cross- over fashion.

#### **4.2.3 Maximal oxygen consumption**

The participants reported to the laboratory 7 to 10d prior to the first trial to complete an incremental maximal exercise test to exhaustion to determine their maximal oxygen uptake ( $\dot{V}O_{2\text{max}}$ ) and maximal heart rate ( $HR_{\text{max}}$ ). All testing took place on the same SRM at the same time of day in the laboratory. Expired air was measured breath- by- breath using an automated open- circuit gas analysis system. The equipment and procedure for this maximal test has previously been described in chapter 2 (pg 53).

#### **4.2.4 Experimental Protocol**

On day 1, the participants reported to the laboratory at 09:00h following an overnight fast. Participants were then asked to void before they were weighed in cycling shorts. After sitting for 5min, a baseline venous blood sample and a 5min expired air sample was collected via standard open circuit spirometry (pg 53). A HR monitor (pg 54) was then attached to the participant and following a standardised 5min warm up at 50W, the participants began their glycogen depleting protocol. This consisted of 2h of cycling on a Velotron cyclePro© ergometer at a heart rate equivalent to 75%  $HR_{\text{max}}$ , followed by four 30s “all- out” sprints separated by 2min of passive recovery. This intensity and duration has previously been shown to significantly reduce muscle glycogen stores (Burke *et al.*, 2003). Heart rate was monitored every 5min throughout the trial to confirm that the participant was cycling at 75%  $HR_{\text{max}}$ . A final venous blood sample and 5min

expired air sample was collected after the cessation of exercise. Participants were then provided with their prescribed recovery diets, beginning with breakfast, 30min after the cessation of exercise. After the participants had consumed their prescribed breakfast they were free to leave the laboratory with their 24h recovery meal plans, which they were asked to follow.

On day two, the participants returned to the laboratory, following the 24h recovery period, 2-3h postprandial. After the participant had been sitting for 5min, a basal venous blood sample and a 5min resting expired air sample were collected as described previously, after which the HR monitor was then attached.

Following a standardised 5min warm up at 50W the participants were required to complete the 40km TT in as short a time as possible. Five minute expired air samples were collected during the last 5min of every 20min time period and venous blood samples were collected in the last min of every 20min time period throughout the TT. The last expired air sample was collected during the last 3km of cycling and the last venous blood sample was taken immediately on cessation of the trial while the participants were still seated on the Velotron cyclePro©. Ratings of perceived exertion (RPE), using Borg's 6- 20 point scale (Borg, 1982), and HR were also recorded every 15min. After the final seated venous blood sample was obtained and the participants had rested they were then allowed to leave the laboratory.

Participants were provided with water ad libitum during trial 1 and this was matched for the second trial. The participants were not aware of their performance times or HR but they were given a verbal reminder every 15min that they were required to continue until they reached the set distance or the participants reached exhaustion.

#### **4.2.5 Test meals**

Isocaloric test meals consisting of either HGI or LGI foods were provided for each participant following the completion of the glycogen depleting protocol. The recovery meals provided  $8\text{g}\cdot\text{kgbw}^{-1}$ , and were split up into four meals and two snacks to be consumed at specific times during the 24-hour period. Breakfast was provided and consumed in the laboratory 30min after the



completion of the glycogen depleting protocol. The participants then left the lab and were instructed to consume lunch three hours later. The participants were also instructed to consume one snack between lunch and the evening meal, which was to be eaten 8h after the glycogen depleting protocol, and a final snack between 20:00 and 21:00h. The participants were then instructed to consume their fourth meal 2- 3 hours prior to arrival in the laboratory to complete their 40km TT. The foods were carefully chosen so that each diet was matched for protein and fat. The GI values of each test food were taken from Foster- Powell *et al.* (2002) and the GI of the total meal was calculated from the weighted means of the GI values for the component foods (Wolever and Jenkins, 1986). To control for participant compliance, the participants were required to complete a food diary of the 48h prior to the trial and on the day of the trial and were required to sign a contract claiming that they had consumed all the necessary foods they were asked to. The mean caloric, macronutrient and GI content of the foods ingested for the high and low GI diets are shown in Table 4.1.

**Table 4.1** Shows the high and low glycaemic index 24h recovery diets, which were consumed by each participant during the recovery period. This example is for a 70kg male participant.

| 24 h Recovery Diets                      |  |  |   |   |
|--|--|--|---|---|
| Time (after exercise)                    | High GI (GI- 70 <sup>b</sup> )   | Nutrient content   | Low GI (GI- 35 <sup>b</sup> )   | Nutrient content  |
| Meals 1 ( $t = 0h$ ) and 4 ( $t = 21h$ ) | 62g cornflakes <sup>a</sup> ,<br>257mL skim milk,<br>80g white bread,<br>10g flora,<br>20g jam,<br>155ml<br>Lucozade original <sup>a</sup> | 730kcal,<br>139g CHO,<br>9.9g fat,<br>20g protein  | 86g muesli,<br>257mL skim milk,<br>67g apple,<br>103g<br>canned peaches,<br>128g yoghurt,<br>257ml apple juice                        | 732kcal,<br>139g CHO,<br>9.0g fat,<br>23g protein   |
| Meal 2 ( $t = 4h$ )                      | 158g white bread<br>154g turkey breast,<br>50g cheese,<br>40g lettuce,<br>180g banana,<br>200ml Lucozade original <sup>a</sup>             | 1076 kcal,<br>148 g CHO,<br>24g fat,<br>63g protein  | 154g whole wheat pasta,<br>150g turkey breast,<br>50g cheese,<br>40g lettuce,<br>185g pasta sauce,<br>150g pear,<br>150mL apple juice | 1075kcal,<br>149g CHO,<br>25g fat,<br>60g protein   |
| Meal 3 ( $t = 8h$ )                      | 255g baked potato,<br>410g canned spaghetti,<br>50g cheese,<br>40g lettuce,<br>67g Mars candy bar,<br>170ml Lucozade Original <sup>a</sup> | 1100kcal,<br>176g CHO,<br>31g fat,<br>28g protein  | 360g chilli beans,<br>200g wheat tortilla,<br>50g cheese,<br>40g lettuce,<br>260mL apple juice  | 1100kcal,<br>176g CHO,<br>29g fat,<br>39g protein   |
| Snacks                                   | 2 x 77g white bread,<br>2 x 20g jam,<br>2 x 10g flora  | 600kcal,<br>96g CHO,<br>17g fat,<br>15g protein  | 170g yoghurt,<br>100g apple,<br>100g flapjack   | 625kcal,<br>97g CHO,<br>25g fat,<br>15g protein   |
| Total Nutrient content                   |  | 3520kcal,<br>560g CHO,<br>84g fat,<br>126g<br>protein<br>(72%CHO,<br>11% fat,<br>17% PRO)<br><b>GI- 70<sup>b</sup></b> |   | 3600kcal,<br>560g CHO,<br>88g fat,<br>135g protein<br>72%CHO,<br>11%fat, 17%<br>PRO)<br><b>GI- 35<sup>b</sup></b> |

Macronutrient breakdown of high GI and low GI test meals provided to each participant calculated from manufacturer's information.

<sup>a</sup>Corn flakes: Kellogg's (UK) Ltd. Manchester UK; Lucozade Original drink: GlaxoSmithKline (UK). <sup>b</sup>Calculated by the method described in Wolever and Jenkins (1986) with GI values taken from Foster-Powell *et al.* (2002).

## Sample collection and analysis

### 4.2.6 Gas analysis

Expired air was measured breath- by- breath using an automated open- circuit gas analysis system.

The equipment employed in gas analysis has previously been described in chapter 2 (pg 53).

#### **4.2.7 Blood analysis**

At each sampling point, one 10mL aliquot of whole blood was taken aseptically from the antecubital vein. A 6ml sample was dispensed into a potassium EDTA Vacuette tube and immediately analysed for whole blood glucose and lactate using equipment described previously (pg 54). The remainder was then dispensed into a serum separator, clot- activator Vacuette tube and immediately centrifuged at 13000g for 10min and analysed for osmolality. The serum was stored at -85°C and later analysed for insulin, FFA and triglycerides. The equipment and techniques involved in the analysis of these hormones and substrates have been previously described in chapter 2 (pg 54).

#### **4.2.8 Data Analysis**

All statistical analyses were conducted using SPSS® for Windows software version 14.0 (SPSS Inc., Chicago, IL, USA). All statistical assumptions were checked and deemed plausible. Sample data are presented as the mean  $\pm$  standard deviation. Repeated measures analysis of variance (ANOVA) on two factors (experimental treatment and time) was used to determine physiological differences between trials. Where the sphericity assumption was violated (as indicated by a significant Mauchly's W), a Huhn-Feldt correction was applied to the degrees of freedom. Sidak-adjusted paired comparisons were performed in the event of a significant F ratio. Time trial performance was analysed using a paired t-test. Two-tailed statistical significance was accepted as  $p < 0.05$ .

### **4.3 Results**

#### *TT performance time*

There was no significance difference between the TT times of the low GI ( $90.7 \pm 11.1$ min) and high GI ( $93.5 \pm 9.29$ min) trials ( $t = 1.1$ ;  $p = 0.35$ ; 95% confidence interval for the mean difference -9.45 to 3.93min).

### *Heart rate and RPE*

There were no significant differences in baseline values between trials for heart rate ( $t = 0.72$ ;  $p = 0.50$ ). There were no significant main effects for trial ( $F = 0.6$ ,  $p = 0.50$ ) or time ( $F = 3.7$ ,  $p = 0.082$ ) and no significant trial-by-time interaction for heart rate ( $F = 1.1$ ,  $p = 0.40$ ). For RPE there was no significant main effect for trial ( $F = 2.3$ ,  $p = 0.19$ ) but there was a significant main effect for time ( $F = 17.3$ ,  $p < 0.001$ ). There also was a significant trial-by-time interaction for RPE ( $F = 5.0$ ,  $p = 0.013$ ), however, no pairwise comparisons between conditions were significant for any time points (Table 4.2). The post- hoc tests for RPE revealed that the time point five (end) was significantly different to time points: 2 ( $p = 0.006$ ), 3 ( $p = 0.009$ ) and 4 ( $p = < 0.001$ ).

**Table 4.2** Shows heart rate and RPE values throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable                     | Trial | Baseline    | 20           | 40           | 60           | End          |
|------------------------------|-------|-------------|--------------|--------------|--------------|--------------|
| HR<br>(b.min <sup>-1</sup> ) | HIGH  |             |              |              |              |              |
|                              | GI    | 56 $\pm$ 10 | 155 $\pm$ 18 | 159 $\pm$ 17 | 158 $\pm$ 18 | 168 $\pm$ 11 |
|                              | LOW   |             |              |              |              |              |
|                              | GI    | 55 $\pm$ 9  | 151 $\pm$ 14 | 156 $\pm$ 12 | 158 $\pm$ 13 | 173 $\pm$ 12 |
| RPE                          | HIGH  |             |              |              |              |              |
|                              | GI    | 6 $\pm$ 0   | 12 $\pm$ 3   | 12 $\pm$ 4   | 12 $\pm$ 4   | 14 $\pm$ 4*  |
|                              | LOW   |             |              |              |              |              |
|                              | GI    | 6 $\pm$ 0   | 14 $\pm$ 2   | 14 $\pm$ 2   | 15 $\pm$ 2   | 17 $\pm$ 2*  |

\* Significantly different ( $P < 0.05$ ) at the end of the TT from all other time points within trials.

### *Substrate Utilization and RER*

There were no significant differences in baseline values between trials for either CHO oxidation ( $t = 1.3$ ;  $p = 0.25$ ) or fat oxidation ( $t = 0.6$ ;  $p = 0.58$ ). There was no significant main effect for time ( $F = 4.0$ ,  $p = 0.09$ ) or trial ( $F = 0.1$ ,  $p = 0.77$ ) for CHO oxidation (Figure 4.1). The trial-by-time interaction for CHO oxidation rates was also not significant ( $F = 1.1$ ,  $p = 0.37$ ). There was a significant main effect for time ( $F = 3.4$ ,  $p = 0.045$ ) but not trial ( $F = 0.1$ ,  $p = 0.76$ ) (Figure 4.2) observed for fat oxidation rates, and no significant trial-by-time interaction ( $F = 1.1$ ,  $p = 0.40$ ). The

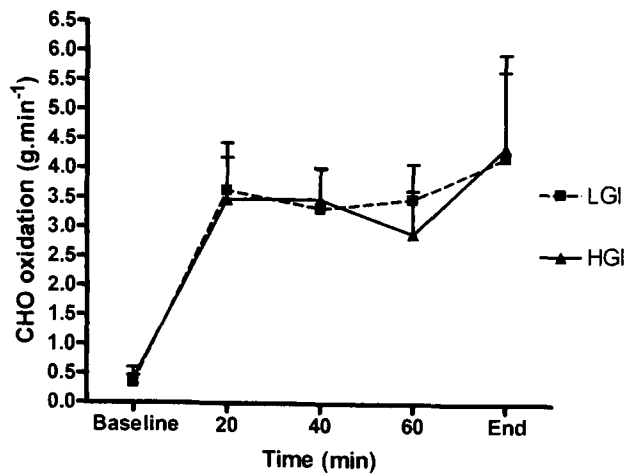
post- hoc tests for fat oxidation revealed that the time point four (60 min) was significantly different to time point 5 ( $p = 0.027$ ). There was no significant main effect for time ( $F = 1.4$ ,  $p = 0.30$ ) or trial ( $F = 1.1$ ,  $p = 0.35$ ) for RER. There was also no significant trial-by-time interaction observed for RER ( $F = 1.1$ ,  $p = 0.35$ ) (Table 4.3).

**Table 4.3** Shows Carbohydrate and Fat oxidation and RER values throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable                            | Trial | Baseline        | 20              | 40              | 60              | End             |
|-------------------------------------|-------|-----------------|-----------------|-----------------|-----------------|-----------------|
| CHO                                 |       |                 |                 |                 |                 |                 |
| Oxidation<br>(g.min <sup>-1</sup> ) | HIGH  |                 |                 |                 |                 |                 |
|                                     | GI    | 0.41 $\pm$ 0.2  | 3.49 $\pm$ 1    | 3.5 $\pm$ 0.5   | 2.92 $\pm$ 0.7  | 4.41 $\pm$ 1.6  |
|                                     | LOW   |                 |                 |                 |                 |                 |
|                                     | GI    | 0.34 $\pm$ 0.1  | 3.64 $\pm$ 0.6  | 3.34 $\pm$ 0.7  | 3.51 $\pm$ 0.6  | 4.23 $\pm$ 1.5  |
| Fat                                 |       |                 |                 |                 |                 |                 |
| Oxidation<br>(g.min <sup>-1</sup> ) | HIGH  |                 |                 |                 |                 |                 |
|                                     | GI    | 0.01 $\pm$ 0.1  | 0.24 $\pm$ 0.3  | 0.26 $\pm$ 0.2  | 0.41 $\pm$ 0.2* | 0.06 $\pm$ 0.4  |
|                                     | LOW   |                 |                 |                 |                 |                 |
|                                     | GI    | 0.02 $\pm$ 0.1  | 0.21 $\pm$ 0.2  | 0.33 $\pm$ 0.3  | 0.22 $\pm$ 0.2* | 0.09 $\pm$ 0.4  |
| RER                                 |       |                 |                 |                 |                 |                 |
|                                     | HIGH  |                 |                 |                 |                 |                 |
|                                     | GI    | 0.97 $\pm$ 0.11 | 0.81 $\pm$ 0.36 | 0.95 $\pm$ 0.04 | 0.92 $\pm$ 0.05 | 0.98 $\pm$ 0.07 |
|                                     | LOW   |                 |                 |                 |                 |                 |
|                                     | GI    | 0.93 $\pm$ 0.07 | 0.96 $\pm$ 0.04 | 0.94 $\pm$ 0.04 | 0.96 $\pm$ 0.03 | 0.97 $\pm$ 0.07 |

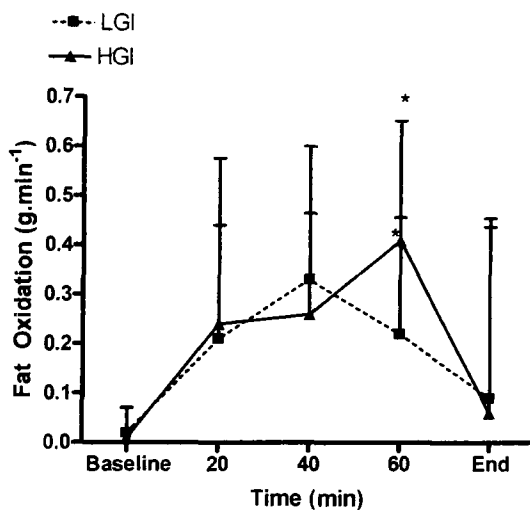
\* Significantly different ( $P < 0.05$ ) at 60 min from the end of TT.

**Figure 4.1** Shows the carbohydrate oxidation throughout the time trial for both the low and high GI recovery diets.



No significant difference in carbohydrate oxidation during exercise between the low and high recovery diets ( $p > 0.05$ ).

**Figure 4.2** Shows the fat oxidation throughout the time trial for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.



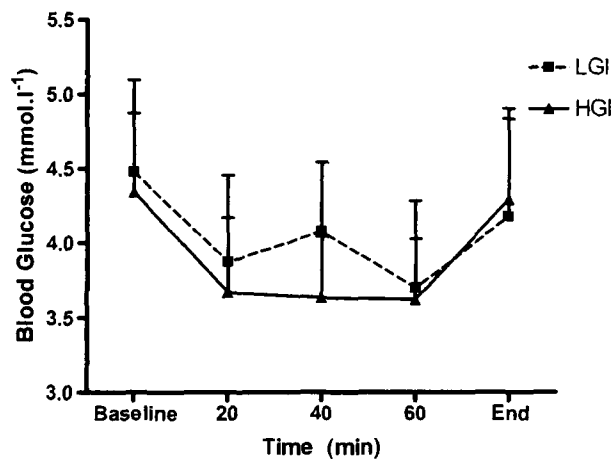
Fat oxidation values were significantly different at 60 min from the End time point for both recovery diets (\* $p < 0.05$ ).

#### *Whole Blood Glucose, Free Fatty Acids (FFA), Triglycerides (TGA)*

There were no significant differences in baseline values between trials for whole blood glucose ( $t = 0.5$ ;  $p = 0.64$ ), FFA ( $t = 1.5$ ;  $p = 0.18$ ), or TGA ( $t = 1.8$ ;  $p = 0.11$ ). For whole blood glucose

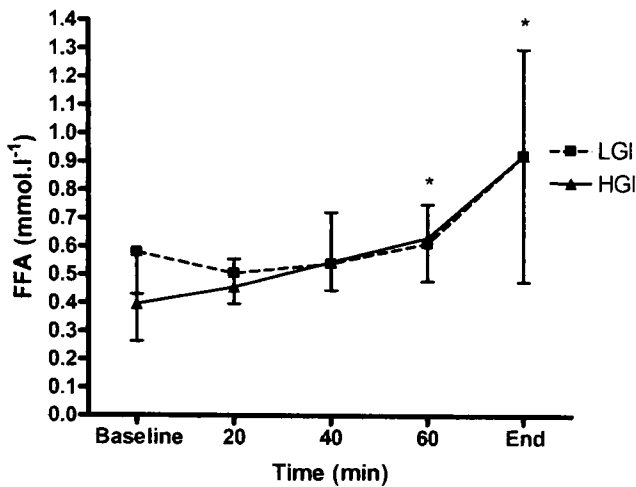
concentration, there was no significant main effects for time ( $F = 3.0$ ,  $p = 0.06$ ) and trial ( $F = 1.3$ ,  $p = 0.30$ ) and no significant trial-by-time interaction ( $F = 0.9$ ,  $p = 0.5$ ) (Figure 4.3). There was a significant main effect for time ( $F = 37.0$ ,  $p = 0.001$ ) but not trial ( $F = 0.003$ ,  $p = 0.96$ ) for FFA (Figure 5.4), and no significant trial-by-time interaction ( $F = 0.2$ ,  $p = 0.71$ ). The post- hoc tests for FFA revealed that the time point four (60 min) was significantly different to time point 2 ( $p = < 0.001$ ). Time point five (end) was also significantly different from time points: 2 ( $p = < 0.001$ ), 3 ( $p = < 0.001$ ) and 4 ( $p = < 0.001$ ). There was no significant main effect for time ( $F = 2.3$ ,  $p = 0.12$ ) or trial ( $F = 1.9$ ,  $p = 0.23$ ) and no significant trial-by-time interaction for TGA ( $F = 0.2$ ,  $p = 0.92$ ) (Table 4.4).

**Figure 4.3** Shows the glucose concentration throughout the time trial for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.



No significant difference in glucose concentration during exercise between the low and high GI recovery diets ( $p > 0.05$ ).

**Figure 4.4** Shows the free fatty acid (FFA) concentration throughout the time trial for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.



FFA values were significantly different at the End from 20, 40 and 60 mins and significantly different at 60 min from 20 min for both recovery diets (\* $p < 0.05$ ).

#### *Serum insulin*

There was no significant difference in baseline values between trials for insulin ( $t = 1.3$ ;  $p = 0.24$ ). There was no significant main effect for time ( $F = 1.4$ ,  $p = 0.28$ ) or trial ( $F = 5.7$ ,  $p = 0.54$ ) for insulin concentration. The trial-by-time interaction for insulin concentration was also not significant ( $F = 0.9$ ,  $p = 0.45$ ) (Table 4.4).

#### *Whole Blood Lactate*

No significant difference was observed between pre-exercise whole blood lactate concentrations for the high GI ( $1.00 \pm 0.33 \text{ mmol.l}^{-1}$ ) and low GI trials ( $1.30 \pm 0.54 \text{ mmol.l}^{-1}$ ) ( $t = 2.2$ ;  $p = 0.075$ ). There was no significant main effect for time ( $F = 2.6$ ,  $p = 0.10$ ) or trial ( $F = 3.4$ ,  $p = 0.14$ ) and no significant trial-by-time interaction for whole blood lactate concentration ( $F = 1.3$ ,  $p = 0.33$ ) Table 4.4).



**Table 4.4** Shows Glucose, Lactate, FFA, TGA and Insulin values throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable                           | Trial | Baseline         | 20             | 40             | 60              | End              |
|------------------------------------|-------|------------------|----------------|----------------|-----------------|------------------|
| Glucose<br>(mmol.l <sup>-1</sup> ) | HIGH  |                  |                |                |                 |                  |
|                                    | GI    | 4.34 $\pm$ 0.5   | 3.67 $\pm$ 0.1 | 3.63 $\pm$ 0.4 | 3.62 $\pm$ 4.1  | 4.3 $\pm$ 0.6    |
|                                    | LOW   |                  |                |                |                 |                  |
|                                    | GI    | 4.48 $\pm$ 0.6   | 3.87 $\pm$ 0.6 | 4.08 $\pm$ 0.5 | 3.70 $\pm$ 0.6  | 4.18 $\pm$ 0.7   |
| Lactate<br>(mmol.l <sup>-1</sup> ) | HIGH  |                  |                |                |                 |                  |
|                                    | GI    | 0.97 $\pm$ 0.3   | 3.91 $\pm$ 1.5 | 2.77 $\pm$ 1.1 | 2.04 $\pm$ 0.4  | 4.29 $\pm$ 0.6   |
|                                    | LOW   |                  |                |                |                 |                  |
|                                    | GI    | 1.28 $\pm$ 0.5   | 2.88 $\pm$ 1.2 | 2.6 $\pm$ 1.2  | 2.0 $\pm$ 0.5   | 3.27 $\pm$ 1.0   |
| FFA<br>(mmol.l <sup>-1</sup> )     | HIGH  |                  |                |                |                 |                  |
|                                    | GI    | 0.4 $\pm$ 0.03   | 0.46 $\pm$ 0.1 | 0.55 $\pm$ 0.2 | 0.64 $\pm$ 0.1* | 0.93 $\pm$ 0.4** |
|                                    | LOW   |                  |                |                |                 |                  |
|                                    | GI    | 0.6 $\pm$ 0.3    | 0.51 $\pm$ 0.1 | 0.54 $\pm$ 0.1 | 0.61 $\pm$ 0.1* | 0.93 $\pm$ 0.5** |
| TGA<br>(mmol.l <sup>-1</sup> )     | HIGH  |                  |                |                |                 |                  |
|                                    | GI    | 0.97 $\pm$ 0.1   | 0.97 $\pm$ 0.1 | 0.95 $\pm$ 0.1 | 0.98 $\pm$ 0.1  | 1.0 $\pm$ 0.2    |
|                                    | LOW   |                  |                |                |                 |                  |
|                                    | GI    | 1.03 $\pm$ 0.1   | 1.21 $\pm$ 0.5 | 1.14 $\pm$ 0.3 | 1.21 $\pm$ 0.5  | 1.0 $\pm$ 0.2    |
| Insulin<br>(ng.ml <sup>-1</sup> )  | HIGH  |                  |                |                |                 |                  |
|                                    | GI    | 16.58 $\pm$ 15.1 | 5 $\pm$ 3.0    | 3.47 $\pm$ 1.8 | 2.65 $\pm$ 0.1  | 2.97 $\pm$ 2.5   |
|                                    | LOW   |                  |                |                |                 |                  |
|                                    | GI    | 11.71 $\pm$ 15.8 | 3.6 $\pm$ 3.5  | 2.64 $\pm$ 1.5 | 2.39 $\pm$ 0.7  | 2.17 $\pm$ 1.2   |

\* Significantly different ( $P < 0.05$ ) at 60 min from the 20 min of TT.

\*\* Significantly different ( $P < 0.05$ ) at End of TT min from 20, 40 and 60 in of TT.

### Osmolality

Pre- and post -TT osmolality values for the LGI were  $299 \pm 5$  and  $297 \pm 5$ , respectively. Pre- and post -TT osmolality values for the HGI were  $299 \pm 6$  and  $296 \pm 5$ , respectively.

## 4.4 Discussion

The main finding of the present study was that the ingestion of a high GI CHO 24h recovery diet following glycogen depleting exercise, had no greater effect on endurance performance than consuming a low GI CHO 24h recovery diet. This finding can be explained by the absence of any significant differences in physiological responses during the time trials that followed the two recovery diets.

It has previously been demonstrated that the provision of high GI foods in the recovery period is associated with a greater rate and amount of muscle glycogen resynthesis compared to the consumption of a low GI recovery diet (Burke *et al.*, 1993; Joszi *et al.*, 1996; Kiens *et al.*, 1990;

Wee *et al.*, 2005). This is due to the large glycaemic and insulinaemic responses elicited by the consumption of high GI foods compared to low GI foods (Burke *et al.*, 1993; Stevenson *et al.*, 2005b). Since glycogen storage is influenced by both insulin and an available supply of glucose, one would expect in the present study, that CHO foods with the higher GI values would enhance post- exercise refuelling of the body's depleted glycogen stores and subsequent athletic performance. However, despite this metabolic profile associated with high GI recovery foods, no differences in metabolic profile or endurance performance were observed between the diets in the present study. Although there was a tendency for fat oxidation and blood glucose concentrations to be greater throughout the low GI trial, none of these differences reached statistical significance.

A limitation of the present study, which may offer some explanation for this lack of difference, is the consideration that the glycaemic and insulinaemic responses to the recovery diets were not measured during the recovery period. Knowing this information may have demonstrated the metabolic effects that each meal was initially having on substrate availability and recovery and provided more information about the initial recovery immediately after the cessation of exercise. It could be that consuming a high GI recovery diet was associated with higher initial rates of glucose availability/ uptake by the muscle, compared to the low GI recovery diet, however, provided enough CHO is consumed in the 24h recovery period, muscle glycogen may have been replenished regardless of the GI of the recovery diet, which would contrast with the findings of Burke *et al.* (1993). However, as this was not measured it was not possible to identify which meal was associated with the greatest initial rates of glycogen recovery. Additionally, this speculation may be supported by a previous study in which, participants were provided with a high GI recovery diet at different times over a 24h period (Parkin *et al.*, 1997). It was found that, providing sufficient CHO is provided and consumed during the recovery period, delaying the consumption of high GI meal by 2 h has no effect on the rate of muscle glycogen synthesis at 8 and 24h post- exercise (Parkin *et al.*, 1997). Therefore, from this, it could be implied in the present study that possibly there was no difference in recovery and performance between the two diets because the provision of CHO may

have been enough for the recovery of depleted glycogen stores regardless of the GI of the CHO foods.

The results of the present study are also similar to the findings of a recent study conducted by Erith *et al.* (2006), who observed no difference in either metabolism or performance after consuming both low GI and high GI recovery diets. Erith *et al.* (2006) placed this lack of difference down to muscle glycogen content between the diets, which was probably different during the exercise test but reduced to similar values at the end of the test due to the nature of the test. A limiting factor in the results of Erith's *et al.* (2006) study may be the type of exercise employed throughout the trial as exercise that has an eccentric component (i.e. running) can result in microscopic damage to muscle fibers (Thompson, Nicholas, and Williams, 1999). This damage is known to stimulate the infiltration of white blood cells, which rely on blood glucose as their sole energy substrate, subsequently resulting in an increased competition for blood glucose and a decreased rate of muscle glycogen synthesis (Doyle *et al.*, 1993).

The findings of the present study do not support previous research by Burke *et al.* (1993) who demonstrated that consuming high GI recovery diets following glycogen depleting exercise resulted in 48% greater muscle glycogen concentration compared to a low GI recovery diet, although this was not assessed in the present study. However, Burke *et al.* (1993) attributed this effect to greater glucose and insulin levels in response to the high GI recovery diet, which although assessed in the present study was not different between the low and high GI recovery diets. Although muscle biopsies and post- prandial blood samples were obtained, exercise performance was not assessed. However, as Erith *et al.* (2006) pointed out, given the high GI recovery diet was associated with greater resting muscle glycogen content, it would be seem reasonable to suggest that the participants would have performed better following the consumption of the high GI recovery meal since the ability to perform moderate- to high- intensity exercise is dependent on muscle glycogen availability.

The present study also contrasts with a more recent study by Stevenson *et al* (2005a) who demonstrated that the consumption of a low GI 24h recovery diet, following glycogen depleting

exercise, resulted in favourable alterations in metabolism and a greater endurance capacity during steady state exercise compared to a high GI recovery diet. The improvement in performance in this study was attributed to greater fat oxidation rates and FFA availability, providing a glycogen sparing effect, which at 60 min into our trial, was the same. Erith *et al.* (2006) suggested, that the runners in Stevenson's *et al* (2005a) study probably started the second run with higher muscle glycogen stores, however, it is likely that they would have used more glycogen during the run than if they had consumed the low GI meal. Therefore, it was speculated that after approximately an hour of running, the muscle glycogen content might have been similar between the diets even though the initial rates of glycogenolysis were different. Although Stevenson's *et al* (2005a) study employed a protocol which has been shown to reduce muscle glycogen, the participant's were required to exercise in the fasted state the following day, which would have altered and shown changes in metabolism anyway. This does not mean that the improvement in performance was because of improved recovery following the consumption of low GI foods, but that in the fasted state more fuel was available after the ingestion of low GI carbohydrates, which is not typical of athletic performance. Furthermore, when conditions representative of a typical athletic performance have been followed, i.e. a regular pre- exercise meal ingested, no consistent beneficial effects on performance have been observed.

#### **4.5 Conclusion**

In conclusion, the results of this study suggest that although consuming a low GI recovery diet is associated with greater resting glucose levels and a greater CHO oxidation towards the end of exercise, this appears to have no further benefit to performance, providing the amount of CHO consumed is enough to replenish glycogen depleted muscles. Given the controversy within this area, further, more invasive research is warranted into the direct effect the glycaemic index of recovery meals can have on muscle glycogen resynthesis and subsequent endurance performance.

## Chapter 5

### **The effects of high and low GI recovery meals on substrate utilisation and TT performance following a short- term recovery period**

#### **5.1 Introduction**

The capacity to perform physical exercise of a moderate to high intensity is related to muscle glycogen availability at the onset of exercise (Bergstrom *et al.*, 1967; Coyle *et al.*, 1986). When athletes are undertaking prolonged training or competition sessions, the provision of extra CHO is essential for the optimization of fuel availability and to promote muscle glycogen resynthesis post-exercise, in order to maximize endogenous CHO stores for the subsequent training session. Therefore, the restoration of muscle glycogen content during the post exercise recovery period is a major challenge for athletes who have busy training and competition schedules (Burke *et al.*, 1993).

Muscle glycogen resynthesis following moderate-to-high intensity exercise, primarily depends on CHO intake following exercise (Robergs, 1991). The rate of muscle glycogen storage in glycogen depleted muscle has been shown to be higher ( $7.7\text{mmol.kgww.h}^{-1}$ ) for the first 2h period when the participants ingested CHO immediately post exercise but lower ( $4\text{mmol.kgww.h}^{-1}$ ) when CHO consumption was delayed for 2h after the cessation of exercise (Ivy *et al.*, 1988). This is because early post- exercise recovery is associated with an exercise- induced permeability of the muscle cell membrane to glucose and an increase in muscle insulin sensitivity (Burke, Collier, Hargreaves, 1998). Immediate CHO feeding takes advantage of these effects, slightly increasing glycogen storage rates and is most important when the interval between exercise sessions is short and the rate of refuelling must be maximized (Ivy *et al.*, 1988).

However, while the benefits of high CHO recovery diets are clearly recognized little attention has been given to the possible influences of the different types of carbohydrates on subsequent performance. Since glycogen storage is influenced by both insulin and a rapid supply of glucose substrate, research has been conducted into the effects of the GI of CHO supplements on

the restoration of muscle glycogen during post- exercise recovery (Burke *et al.*, 1993; Stevenson *et al.*, 2005). Due to the large glycaemic and insulinaemic responses following the ingestion of high GI foods (Erith *et al.*, 2006), which favour muscle glycogen resynthesis, these types of foods are generally recommended to athletes, during recovery from exercise. In contrast, the consumption of low GI foods has repeatedly been shown to produce lower glycaemic and insulinaemic responses during rest in the postprandial period compared to high GI foods (Stevenson *et al.*, 2005). However, research into the effects of differing GI of foods on recovery and performance is equivocal as inconsistent findings have been reported.

Burke *et al.* (1993) for example, showed that consuming high GI meals during a 24h recovery period from prolonged heavy exercise resulted in higher muscle glycogen resynthesis than following the consumption of an iso- caloric low GI diet. It would be reasonable to assume that endurance capacity would be greater during subsequent exercise, however, this was not investigated. Later, Kiens *et al.* (1990) found high GI recovery meals to be associated with greater muscle glycogen resynthesis and improved subsequent athletic performance compared to the consumption of low GI recovery meals. According to these studies, high GI foods seem to be the desirable CHO supplementation during the post-exercise recovery period as they elicit greater substrate availability for glycogen re-synthesis. In contrast to previous findings however, Stevenson *et al.* (2005) provided their participants with low and high GI 24h recovery diets prior to performing an exercise to exhaustion protocol. The main finding of this study (Stevenson *et al.*, 2005) was that the ingestion of a low GI recovery diet consumed in the 24h recovery period following glycogen depleting exercise, resulted in a greater endurance capacity during steady state exercise in the post-absorptive state compared with a high GI CHO diet.

Research has been conducted into the effects of the ingestion of foods of different GI before exercise (Thomas *et al.*, 1991; 1994) and during 24hour recovery periods following glycogen depleting exercise (Stevenson *et al.*, 2005). Little research however, has been conducted into the effects of the GI of meals on shorter recovery periods between multiple bouts of exercise (Erith *et al.*, 2006) and further research is needed to investigate the influence of the GI of foods on post-

exercise recovery (Siu and Wong, 2004). Additionally, research into the effects of the GI of CHO feedings during 24h recovery is limited and the results of studies investigating muscle glycogen resynthesis are equivocal. Also, many of the previous studies in this area have provided single foods in the hour before exercise, which is unlikely to reflect normal dietary behaviour and athletes are typically recommended to consume a high CHO meal 3-4hours before exercise, and many recreational athletes will exercise at lunchtime 3-4h after consuming breakfast (Stevenson *et al.*, 2005). Therefore the aim of the present study was to investigate the influence of high and low GI recovery meals on substrate utilisation during a short- term (3h) recovery period and subsequent 5k time trial (TT) performance.

## **5.2 Methods**

### **5.2.1 Participants**

Seven well- trained male cyclists participated in the study. Their mean ( $\pm$  SD) age, height, body weight, and maximum workload ( $W_{\max}$ ) were  $29 \pm 9$ y,  $175.8 \pm 8.9$ cm,  $75.2 \pm 10.2$ kg, and  $310 \pm 48.1$ W, respectively. Maximal workload was measured in order to calculate their glycogen depleting protocol, which is described below.

### **5.2.2 Experimental design**

Each participant performed two experimental trials, each separated by at least seven days and undertaken in a randomised, cross-over manner. During each trial, the participants completed a glycogen- depleting exercise protocol, followed by a 3h recovery period, in which participants immediately consumed either a high GI or low GI CHO test meals providing  $2\text{g}\cdot\text{kgbw}^{-1}$  of CHO. The post-exercise test meals were consumed while the participants were seated in the laboratory. Following the recovery period, the participants then performed a 5km TT, after which, they were free to leave the laboratory.

### 5.2.3 Maximal Workload ( $W_{\max}$ )

One week prior to the actual experiment the participants reported to the laboratory for determination of their maximal workload ( $W$ ) attained ( $W_{\max}$ ) on an SRM (pg 52). The exercise started at 100W for 5min, increasing by 50W every 2.5min. Once the participants reached a heart rate of  $150\text{b}\cdot\text{min}^{-1}$ , the intensity increased by 25W. The participants maintained the revolution rate between 75 and 90rpm. In each individual  $W_{\max}$  was calculated from  $W_{\max} = W_{\text{out}} + (t / 150) \times AW$ , where  $W_{\text{out}}$  is the highest workload that the participant completed,  $t$  is the number of seconds the final uncompleted workload was sustained, and  $AW$  is the workrate increment. The test was terminated when the participant could not maintain a pedal cadence of at least 50rpm.

### 5.2.4 Experimental Protocol

Each participant reported to the laboratory 3h post- prandial and after sitting for 5min, a resting finger prick blood sample was collected and a 5min resting expired air sample was also collected using the Quark b<sup>2</sup> (pg 53). Following a standardised 5min warm- up at 50W, the participants then performed a glycogen depleting exercise protocol on the SRM. This protocol consisted of 2min bouts at 90%  $W_{\max}$ , interspersed with 2min at 50%  $W_{\max}$ . If the participants were unable to complete the 2min intensive exercise at 90%  $W_{\max}$ , the workload was lowered, subsequently to 80% and 70%  $W_{\max}$ . The exercise stopped when the 2min at 70%  $W_{\max}$  could not be completed. This exercise protocol has been initially described by Kuipers *et al.* (1987) and has been used extensively to induce muscle glycogen depletion (e.g. Wallis *et al.*, 2008; Jentjens *et al.*, 2001, van Hall *et al.*, 2000; van Loon *et al.*, 2000). Heart rate (pg 54) was collected continuously during the protocol, and a finger prick blood sample was collected immediately following termination of the trial, while participants were still seated on the SRM. Following the completion of this protocol, the participants were immediately provided with either a high or low GI CHO recovery meal. A 15min post-prandial venous blood sample was then collected and additional venous blood and expired air samples were collected at 30min stages until the onset of the next exercise bout. Following a 3h recovery period, where the participants remained seated quietly in the lab, and following a similar



pre-competition warm up, consisting of approximately 5min cycling at 50W, the participants then performed a 5km TT on the SRM cycle ergometer. Throughout the TT, expired air was collected continuously and a venous blood sample was collected immediately following termination of the TT while participants were still seated on the cycle ergometer. Heart rate was measured continuously throughout the TT, whereas ratings of perceived exertion (RPE), using Borg's 6- 20 point scale (Borg, 1983), were recorded every 2min. Participants were not aware of their performance times and heart rate.

### **5.2.5 Test meals**

The high and low GI meals were consumed in the laboratory immediately following the cessation of exercise. The participants had 15min to consume the test meals and were unaware of the type of meal that they had consumed, until after completion of the study. The high GI meal consisted of Cornflakes (Kelloggs Co., Manchester, UK) and semi-skimmed milk (GI: 72) whereas the low GI meal consisted of Muesli (Kelloggs Co., Manchester, UK) and semi-skimmed milk (GI: 40). The GI values of each test food were taken from Foster- Powell *et al.* (2002), and the GI of the total meal was calculated from the weighted means of the GI values for the component foods (Wolever and Jenkins, 1986). The diets were as closely matched in terms of macronutrients and energy as possible; however, there is a 100 kcal difference, of which it is not immediately apparent where this difference has come from. The test meals are presented in Table 5.1.

**Table 5.1**

Characteristics of the high glycaemic index (GI) and low GI test meals for a 70kg participant providing  $2\text{g.kgbw}^{-1}$  of CHO.

|              |                  | High- GI breakfast   | Low-GI breakfast   |
|--------------|------------------|--|--|
| Description  |                  | 136g Cornflakes,<br>566ml semi-skimmed<br>milk, 368ml water. | 181g Branflakes, 451ml<br>semi-skimmed milk,<br>313ml water. |
| Meal content | Energy (kcal)    | 774  | 672  |
|              | Carbohydrate (g) | 140  | 140  |
|              | Fat (g)          | 12   | 13   |
|              | Protein (g)      | 31   | 27   |
|              | Fibre (g)        | 4  | 11   |
|              | Glycaemic index  | 72*  | 40*  |

Macronutrient breakdown of high GI and low GI test meals provided to each participant calculated from manufacturer's information. \*Calculated by the method of Wolever and Jenkins (1986) Glycaemic index values from Foster- Powell *et al.* (2002).

### ***Sample collection and analysis***

#### **5.2.6 Gas analysis**

Expired air was measured breath- by- breath using an automated open- circuit gas analysis system.

The equipment employed in gas analysis has previously been described in chapter 2 (pg 53).

#### **5.2.7 Blood analysis**

At each sampling point, 12ml of whole blood was taken aseptically from the antecubital vein and dispensed immediately into two different vacutainers. A 6ml sample was dispensed into a potassium EDTA Vacuette tube and immediately analysed for whole blood glucose and lactate using equipment previously described (pg 54). The remainder was dispensed into a serum separator, clot-activator Vacuette tube and was immediately centrifuged at 13000g for 10min and analysed for osmolality. The serum was then stored at  $-85^{\circ}\text{C}$  and later analysed for insulin, FFA and triglycerides. The equipment and procedure involved in the analysis of these hormones and substrates have been previously described in chapter 2 (pg 54).

### 5.2.8 Data Analysis

All statistical analyses were conducted using SPSS® for Windows software version 14.0 (SPSS Inc., Chicago, IL, USA). All statistical assumptions were checked and deemed plausible. Sample data are presented as the mean  $\pm$  standard deviation. Repeated measures analysis of variance (ANOVA) on two factors (experimental treatment and time) was used to determine physiological differences between trials. Where the sphericity assumption was violated (as indicated by a significant Mauchly's W), a Huynh-Feldt correction was applied to the degrees of freedom. Sidak-adjusted paired comparisons were performed in the event of a significant F ratio. Time trial performance was analysed using a paired t-test. Two-tailed statistical significance was accepted as  $p < 0.05$ .

### 5.3 Results

#### *TT performance time*

There was no significance difference between the TT times of the low GI ( $8.5 \pm 1.4\text{min}$ ) and high GI ( $8.1 \pm .9\text{min}$ ) trials ( $t = 1.4$ ;  $p = 0.20$ ; 95% confidence interval for the mean difference -1.06 to 0.28 min).

#### *Substrate Utilization and RER*

There was a significant main effect for time ( $F = 124.8$ ,  $p = < 0.001$ ) and trial ( $F = 8.9$ ,  $p = 0.025$ ) observed for CHO oxidation rates. A significant trial-by-time interaction ( $F = 6.4$ ,  $p = 0.029$ ) was also observed with CHO oxidation rates being significantly higher in the high GI trial at the 150 min recovery time point ( $p = 0.025$ ) and at the end of the TT ( $p = 0.032$ ) compared to the low GI trial (Figure 5.1). The post- hoc tests for CHO oxidation revealed that the time point six (end of TT) was significantly different to time points: 1 ( $p = < 0.001$ ), 2 ( $p = < 0.001$ ), 3 ( $p = < 0.001$ ), 4 ( $p = < 0.001$ ), and 5 ( $p = < 0.001$ ). There was a significant main effect for time ( $F = 21.0$ ,  $p = 0.001$ ) and trial ( $F = 9.6$ ,  $p = 0.021$ ) for fat oxidation. There was no significant trial-by-time interaction observed for fat oxidation ( $F = 4.5$ ,  $p = 0.052$ ). The post- hoc tests for fat oxidation revealed that time point six (end of TT) was significantly different to time points: 1 ( $p = 0.022$ ), 2 ( $p = 0.043$ ), 4

( $p = 0.046$ ), and 5 ( $p = 0.046$ ). There was a significant main effect for time ( $F = 44.2$ ,  $p < 0.001$ ) but not trial ( $F = 1.1$ ,  $p = 0.34$ ) observed for RER, and no significant trial-by-time interaction ( $F = 0.9$ ,  $p = 0.49$ ). ). The post- hoc tests for RER revealed that the time point six (end of TT) was significantly different to time points: 1 ( $p = 0.005$ ), 2 ( $p = 0.002$ ), 3 ( $p = 0.006$ ), 4 ( $p = 0.004$ ), and 5 ( $p = 0.002$ ) (Table 5.2).

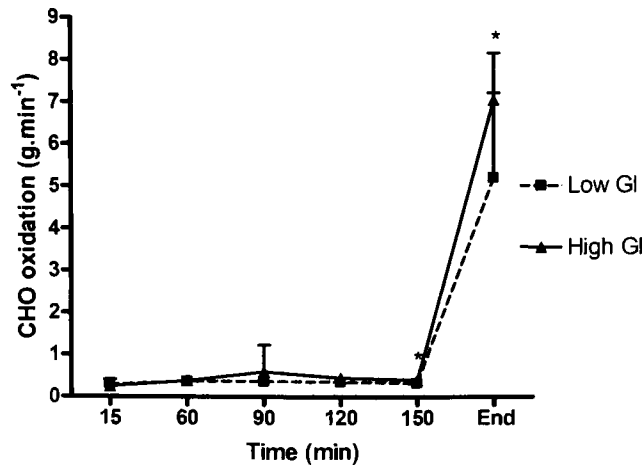
**Table 5.2** Shows CHO and Fat Oxidation and RER values throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable  | Trial | 15              | 60              | 90              | 120                 | 150                | End               |
|---|-------|-----------------|-----------------|-----------------|---------------------|--------------------|-------------------|
| CHO   |       |                 |                 |                 |                     |                    |                   |
| Oxidation<br>( $\text{g} \cdot \text{min}^{-1}$ ) | HIGH  |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.25 \pm 0.2$  | $0.38 \pm 0.1$  | $0.6 \pm 0.6$   | $0.45 \pm 0.1^{**}$ | $0.4 \pm 0.1^{**}$ | $7.07 \pm 1.1^*$  |
|   | LOW   |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.29 \pm 0.1$  | $0.37 \pm 0.1$  | $0.37 \pm 0.1$  | $0.35 \pm 0.03$     | $0.32 \pm 0.1$     | $5.22 \pm 2.0^*$  |
| Fat   |       |                 |                 |                 |                     |                    |                   |
| Oxidation<br>( $\text{g} \cdot \text{min}^{-1}$ ) | HIGH  |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.12 \pm 0.1$  | $0.07 \pm 0.04$ | $0.04 \pm 0.3$  | $0.05 \pm 0.1$      | $0.06 \pm 0.02$    | $0.83 \pm 0.2^*$  |
|   | LOW   |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.11 \pm 0.04$ | $0.08 \pm 0.03$ | $0.08 \pm 0.03$ | $0.08 \pm 0.02$     | $0.09 \pm 0.3$     | $0.25 \pm 0.6^*$  |
| RER   | HIGH  |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.83 \pm 0.1$  | $0.9 \pm 0.06$  | $0.92 \pm 0.1$  | $0.93 \pm 0.04$     | $0.91 \pm 0.02$    | $1.13 \pm 0.03^*$ |
|   | LOW   |                 |                 |                 |                     |                    |                   |
|   | GI    | $0.85 \pm 0.1$  | $0.89 \pm 0.04$ | $0.89 \pm 0.04$ | $0.89 \pm 0.02$     | $0.87 \pm 0.04$    | $1.13 \pm 0.1^*$  |

\* Significantly different ( $P < 0.05$ ) at the end of the TT from all other time points.

\* Significantly different ( $P < 0.05$ ) in high GI than low GI at 120 and 150 min.

**Figure 5.1** Carbohydrate oxidation throughout the 3h postprandial period and the 5km time trial in the low glycaemic index (GI) and high GI conditions. Data are presented as means  $\pm$  SD.

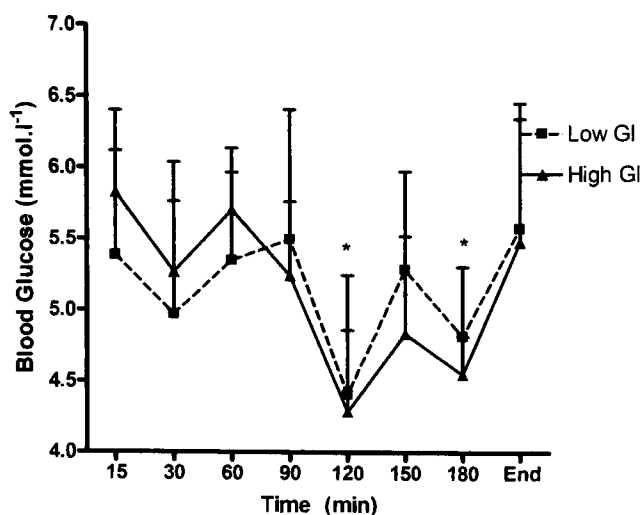


\* Significantly higher in the high GI trial at 150 min and the end of the TT ( $p < 0.05$ ).

#### *Whole Blood Glucose, Free Fatty Acids (FFA), Triglycerides (TGA)*

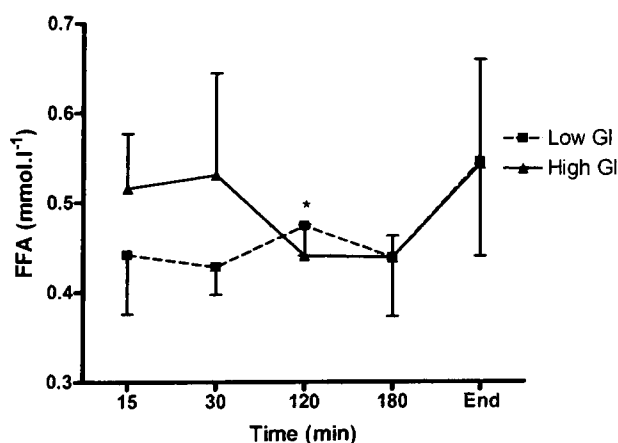
There were no significant differences in baseline values between trials for whole blood glucose ( $t = 0.8$ ;  $p = 0.46$ ). For whole blood glucose concentration, there was a significant main effect for time ( $F = 7.9$ ,  $p < 0.001$ ) but not trial ( $F = 0.005$ ,  $p = 0.94$ ) and no significant trial-by-time interaction ( $F = 1.18$ ,  $p = 0.33$ ) (Figure 5.2). ). The post-hoc tests for blood glucose revealed that the time point five (120 min) was significantly different to time points: 1 ( $p = 0.007$ ), 2 ( $p = 0.048$ ), 3 ( $p = 0.004$ ), 4 ( $p = 0.001$ ), and time point seven (180 min) was significantly different to time point three ( $p = 0.035$ ). There was a significant main effect for time ( $F = 3.2$ ,  $p = 0.029$ ) but not trial ( $F = 5.4$ ,  $p = 0.059$ ) for FFA (Figure 5.3). There was also a significant trial-by-time interaction ( $F = 2.9$ ,  $p = 0.044$ ) for FFA concentration, with the low GI meal having significantly greater values ( $0.47 \pm 0.03 \text{ mmol.l}^{-1}$ ) at 120min into recovery ( $p = 0.043$ ) compared to the high GI meal ( $0.44 \pm 0.04 \text{ mmol.l}^{-1}$ ). There was no significant main effect for time ( $F = 1.5$ ,  $p = 0.26$ ) or trial ( $F = 2.4$ ,  $p = 0.17$ ) and no significant trial-by-time interaction for TGA ( $F = 1.4$ ,  $p = 0.28$ ).

**Figure 5.2** Blood glucose concentration during the 3h postprandial period and throughout the 5km time trial in the low glycaemic index (GI) and high GI conditions. Data are presented as means  $\pm$  SD.



\* = Significantly different at 120 min to 15, 30, 60 and 90 min.  
Significantly different at 180 min to 60 min ( $p < 0.05$ ).

**Figure 5.3** Free fatty acid concentration during the 3h postprandial period and throughout the 5km time trial in the low glycaemic index (GI) and high GI conditions. Data are presented as means  $\pm$  SD.



\* Significantly higher in the low GI trial compared to the high GI trial at 120 min ( $p < 0.05$ ).

### Serum insulin

There was a significant main effect for time ( $F = 8.7$ ,  $p < 0.001$ ) but not trial ( $F = 3.7$ ,  $p = 0.11$ ) for insulin concentration. The trial-by-time interaction for insulin concentration was also not significant

( $F = 0.8$ ,  $p = 0.47$ ). ). The post- hoc tests for insulin concentrations revealed that the time point two (30 min) was significantly different to time points: 4 ( $p = 0.003$ ) and 5 ( $p = 0.016$ ).

**Table 5.3** Shows FFA, TGA and Insulin values throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable                          | Trial | 15               | 30              | 120               | 180              | End              |
|-----------------------------------|-------|------------------|-----------------|-------------------|------------------|------------------|
| FFA<br>(mmol.l <sup>-1</sup> )    | HIGH  |                  |                 |                   |                  |                  |
|                                   | GI    | 0.52 $\pm$ 0.06  | 0.53 $\pm$ 0.1  | 0.44 $\pm$ 0.04   | 0.44 $\pm$ 0.02  | 0.54 $\pm$ 0.1   |
|                                   | LOW   |                  |                 |                   |                  |                  |
|                                   | GI    | 0.44 $\pm$ 0.1   | 0.42 $\pm$ 0.03 | 0.47 $\pm$ 0.03** | 0.44 $\pm$ 0.1   | 0.55 $\pm$ 0.1   |
| TGA<br>(mmol.l <sup>-1</sup> )    | HIGH  |                  |                 |                   |                  |                  |
|                                   | GI    | 1.23 $\pm$ 0.3   | 1.68 $\pm$ 0.6  | 1.16 $\pm$ 0.4    | 0.44 $\pm$ 0.02  | 144 $\pm$ 0.3    |
|                                   | LOW   |                  |                 |                   |                  |                  |
|                                   | GI    | 0.97 $\pm$ 0.3   | 1.44 $\pm$ 0.7  | 1.26 $\pm$ 0.2    | 1.03 $\pm$ 0.3   | 1.32 $\pm$ 0.2   |
| Insulin<br>(ng.ml <sup>-1</sup> ) | HIGH  |                  |                 |                   |                  |                  |
|                                   | GI    | 40.34 $\pm$ 23.9 | 50 $\pm$ 20.4*  | 30.7 $\pm$ 22.3   | 22.48 $\pm$ 12.6 | 27.11 $\pm$ 10.5 |
|                                   | LOW   |                  |                 |                   |                  |                  |
|                                   | GI    | 23.74 $\pm$ 6.4  | 34.1 $\pm$ 9.2* | 25.11 $\pm$ 16.5  | 20.51 $\pm$ 10.4 | 19.27 $\pm$ 11.8 |

\*\* Significantly different ( $P < 0.05$ ) in the low GI at 120 min compared to high GI.

\* Significantly different ( $P < 0.05$ ) at 30 min within trial than at 180 min and end time points.

### *Whole Blood Lactate*

No significant difference was observed between pre-exercise whole blood lactate concentrations for the high GI ( $1.10 \pm 0.46$ mmol.l<sup>-1</sup>) and low GI trials ( $1.13 \pm 0.21$ mmol.l<sup>-1</sup>) ( $t = -0.2$ ;  $p = 0.87$ ). There was a significant main effect for time ( $F = 126.2$ ,  $p < 0.001$ ) but not trial ( $F = 2.9$ ,  $p = 0.14$ ) and no significant trial-by-time interaction for whole blood lactate concentration ( $F = 0.4$ ,  $p = 0.91$ ). The post- hoc tests for lactate concentration revealed that time point six (150 min) was significantly different to time points: 1 ( $p = 0.006$ ), 2 ( $p = 0.002$ ), 3 ( $p = < 0.001$ ), 4 ( $p = < 0.001$ ) and 5 ( $p = < 0.001$ ). Time point seven (180 min) was significantly different to time points: 1 ( $p = 0.001$ ), 2 ( $p = < 0.001$ ), 3 ( $p = 0.001$ ) 4 ( $p = < 0.001$ ), and 5 ( $p = < 0.001$ ). Time point eight (end of TT) was also significantly different from time points: 1 ( $p = < 0.001$ ), 2 ( $p = < 0.001$ ), 3 ( $p = < 0.001$ ), 4 ( $p = < 0.001$ ), and 5 ( $p = < 0.001$ ).

**Table 5.4** Shows Glucose and Lactate concentration throughout the TT for both the low and high GI recovery diets. Data are presented as means  $\pm$  SD.

| Variable                        | Trial   | 15             | 30             | 60             | 90             | 120              | 150             | 180              | End              |
|---------------------------------|---------|----------------|----------------|----------------|----------------|------------------|-----------------|------------------|------------------|
| Glucose (mmol.l <sup>-1</sup> ) | HIGH GI | 5.82 $\pm$ 0.6 | 5.27 $\pm$ 0.8 | 5.71 $\pm$ 0.4 | 5.25 $\pm$ 0.5 | 4.29 $\pm$ 0.6** | 4.84 $\pm$ 0.7  | 4.55 $\pm$ 0.3** | 5.49 $\pm$ 0.9   |
|                                 | LOW GI  | 5.38 $\pm$ 0.7 | 4.97 $\pm$ 0.8 | 5.35 $\pm$ 0.6 | 5.5 $\pm$ 0.9  | 4.41 $\pm$ 0.8** | 5.29 $\pm$ 0.7  | 4.82 $\pm$ 0.5** | 5.59 $\pm$ 0.9   |
| Lactate (mmol.l <sup>-1</sup> ) | HIGH GI | 1.71 $\pm$ 0.5 | 1.55 $\pm$ 0.4 | 1.76 $\pm$ 0.7 | 1.53 $\pm$ 0.5 | 0.97 $\pm$ 0.2   | 1.31 $\pm$ 0.3* | 0.93 $\pm$ 0.3   | 10.01 $\pm$ 2.2* |
|                                 |         | 2.25 $\pm$ 0.8 | 1.97 $\pm$ 0.6 | 1.87 $\pm$ 0.8 | 1.71 $\pm$ 0.5 | 1.12 $\pm$ 0.4   | 1.39 $\pm$ 0.4* | 0.99 $\pm$ 0.3   | 10.26 $\pm$ 2.6* |

\* Significantly different ( $P < 0.05$ ), within trials, at 150 min and the end of TT from time points 1-5.

\*\* Significantly different ( $P < 0.05$ ), within trials, at 120 min from time points 1-4 and at 180 min to time point 3.

### Osmolality

Pre- and post -TT osmolality values for the LGI were  $297 \pm 3$  and  $296 \pm 5$ , respectively. Pre- and post -TT osmolality values for the HGI were  $295 \pm 2$  and  $293 \pm 7$ , respectively.

## 5.4 Discussion

The aim of this study was to investigate whether consuming either a high or low GI recovery meal was associated with favourable changes in physiological responses and a subsequent improvement in time trial ergometer performance, following a short- term recovery period.

Typically high GI meals, due to their large glycaemic and insulinaemic responses have been associated with greater rates of muscle glycogen resynthesis and a subsequent improvement in performance (Stevenson *et al.*, 2005). The present study, however, failed to confirm any differences in TT performance between low and high GI recovery meals despite significant changes in substrate availability and utilisation between the high and low GI meals. During the postprandial period there was no significant difference in substrate oxidation, which supports previous research (Wee *et al.*, 1999; Wu and Williams, 2006). However, towards the end of the postprandial period (150min) and throughout the TT the high GI recovery meal was associated with significantly greater CHO oxidation rates compared to the low GI recovery meal. In contrast to previous research by Moore *et al.* (2009), compared with a high GI meal, ingestion of a low GI meal before exercise resulted in significantly less CHO oxidation and a greater fat oxidation towards the end of the postprandial



period (150min) and during the TT. In the present study, both the high and low GI meals provided 140 g CHO. Previous studies using foods of GI similar to the high GI meal, providing 2- 2.5g.kgBW<sup>-1</sup> of CHO, have reported an increase in muscle glycogen content of between 10 and 40% over a 3-4h postprandial period (Chryssanthopoulos, 1995; Coyle *et al.*, 1985; Neufer *et al.*, 1987). Therefore, the increased CHO oxidation in the present study may provide some support for this speculation. Coyle *et al.* (1985) reported that compared with the fasted state, increased CHO oxidation during exercise paralleled the elevated pre- exercise muscle glycogen concentration after a high GI pre- exercise CHO meal. It could be that, although not assessed in the present study, the high GI recovery meal was associated with greater rates of muscle glycogen resynthesis as indicated by the increased CHO oxidation rates. In contrast, a greater portion of the un- oxidized CHO from the low GI meal may have remained in the gut at the onset of exercise (Thorne, Thompson, Jenkins, 1983).

Additionally, it has previously been reported that, when a low GI breakfast is consumed 3h before exercise, less CHO is stored as muscle glycogen than when a high GI breakfast is consumed (Wee *et al.*, 2005). This 15% increase in muscle glycogen concentration was reported at the end of a 3h postprandial period after the high GI breakfast. However, only a small non- significant increase in muscle glycogen was reported after the low GI breakfast. This was accounted for primarily by the low glycaemic and insulinaemic responses to the low GI meal, followed by the slow digestion and absorption of the ingested foods. As similar breakfast cereals and postprandial period was measured in the study by Wee *et al.* (2005) as in the present one, it could be inferred that in the present study, muscle glycogen may have increased by similar amounts, if not more in the high GI trial, due to the glycogen depletion protocol.

In the study of Wee *et al.* (2005), however, it was found that although the low GI meal contributed less CHO to muscle glycogen synthesis in the postprandial period, a sparing of muscle glycogen during subsequent exercise was observed in the low GI trial, which was most likely a result of better maintained fat oxidation. Similarly, in the present study, the low GI trial was associated with higher fat oxidation rates ( $0.09 \pm 0.03\text{g}\cdot\text{min}^{-1}$ ) at 150min into recovery and

throughout the TT compared to the high GI trial ( $0.06 \pm 0.02 \text{ g}\cdot\text{min}^{-1}$ ). Also, FFA concentration was also significantly higher in the low GI trial ( $0.47 \pm 0.03 \text{ mmol}\cdot\text{l}^{-1}$ ) at the 120min point compared to the high GI trial ( $0.44 \pm 0.04 \text{ mmol}\cdot\text{l}^{-1}$ ). Fat oxidation rates may have been higher in the low GI trial as pre-exercise substrate availability is an important regulator of the patterns of fuel oxidation during exercise (Arkinstall *et al.*, 2004). Therefore, it could be that the higher rate of fat oxidation and consequently lower CHO oxidation rate in the low GI trial might be a result of lower pre-exercise muscle glycogen concentrations compared to the high GI trial.

This increased rate of fat oxidation observed in the low GI trial, however may be beneficial for both recovery and subsequent performance. Although it is widely accepted that muscle glycogen is the primary fuel source during prolonged exercise (Romijn *et al.*, 1993), IMTG has been recognised as an important substrate source during prolonged exercise (van Loon, 2004). Several studies have reported a reduction in IMTG concentrations during prolonged exercise (Brechtel *et al.*, 2001; Johnson *et al.*, 2003; van Loon *et al.*, 2003; Krssak *et al.*, 2000). Therefore, the repletion of both muscle glycogen and IMTG stores during the recovery period may need to be taken into consideration. Recent research has reported that a high CHO diet consumed after exercise inhibits post-exercise resynthesis of IMTG because increased FFA concentrations are required for the replenishment of IMTG content (Decombaz *et al.*, 2001; Starling *et al.*, 1997). Also, Kiens and Richter (1998) reported a decrease in IMTG concentration during recovery from glycogen depleting exercise despite a large intake of CHO ( $8 \text{ g}\cdot\text{kgbw}^{-1}$ ). These studies reported that muscle glycogen repletion has such high metabolic priority during recovery that utilization of lipids is essential to cover the energy expenditure in muscle. Therefore, it could be that in the present study, the higher FFA concentration and fat oxidation during both the postprandial period and the TT allowed for the resynthesis of some IMTG as well as the replenishment of muscle glycogen as a result of the CHO intake. This could help to explain the increased fat oxidation and FFA in the low GI trial. This issue, however, warrants further research.

Previously, an improvement in endurance performance has been reported when plasma FFA concentrations have been elevated (Pitsiladis *et al.*, 1999; Lambert *et al.*, 1997) and investigators

have suggested glycogen sparing as the main reason for this improvement (Jeukendrup *et al.*, 1998). Therefore, one may have expected to see an improvement in performance in the present study, however no difference in performance was observed between the low and high GI recovery diets.

Given the observed differences in substrate utilisation and oxidation between the trials, with the high GI recovery meal being associated with greater CHO oxidation and the low GI being associated with greater fat oxidation, one might have expected to see differences in TT performance between the trials. Previous research investigating CHO intake on recovery and performance has typically employed performance measures lasting longer than 1h (Stevenson *et al.*, 2005; Wee *et al.*, 2005). However, in this study the 5km TT employed as the performance measure may not have sufficiently challenged the glycogen stores to see a performance effect between the diets. It may have been that the higher fat oxidation observed in the low GI trial may have spared muscle glycogen during exercise, which, had the TT being of a longer duration, might have resulted in an improvement in endurance performance by delaying the depletion of muscle glycogen. The results of the present study, however, indicate that if the recovery between exercise and training is short and subsequent exercise is not long enough to challenge glycogen stores, then the GI of the recovery meal has no impact on performance provided enough CHO is available during recovery from the last exercise bout.

## **5.5 Conclusion**

The consumption of a high GI recovery meal was associated with significantly greater CHO oxidation rates both towards the end of the postprandial period and throughout the TT. It has previously been suggested that this increase in CHO oxidation may be a reflection of increased muscle glycogen synthesis in the high GI trial compared to the low GI trial (Wee *et al.*, 2005). In contrast, the low GI recovery meal was associated with less CHO oxidation but a greater fat oxidation during the latter stages of recovery and throughout the TT. This increased rate of fat oxidation may be a result of lower pre-exercise muscle glycogen concentrations compared to the high GI trial. Alternatively, the increased fat oxidation may have allowed for the resynthesis of

some IMTG as well as the replenishment of muscle glycogen as a result of the CHO intake. Despite these alterations in substrate utilisation, no differences in TT performance emerged between the two trials. This lack of difference may be due to the 5- km TT performance trial employed, which may not have been long enough to challenge the glycogen stores. The results of this study indicate that following a 3h recovery period, high and low GI recovery meals are associated with greater CHO and fat oxidation rates, respectively, but that if the subsequent exercise is of a short duration, the GI of the recovery diets has no influence on recovery and subsequent exercise performance.

## Chapter 6

### Discussion, practical applications, limitations, and recommendations for future research

#### 6.1 Discussion

The importance of CHO for exercise performance has been recognised since the early classic respiratory exchange studies of Christensen and Hansen (1937) and Bergstrom *et al.* (1967), and since then, considerable attention has been placed on nutritional strategies to maximize endogenous CHO (liver and muscle glycogen), so minimizing the potential effects of CHO depletion (Coyle, *et al.*, 1986). Whilst the importance of adequate dietary CHO for optimal endurance performance has long been recognised (Coggan and Coyle, 1991), the type and amount of CHO might also influence physical performance. Christensen and Hansen (1937) established a link between hypoglycaemia and fatigue by showing that maintenance of euglycemia and CHO oxidation late in exercise can delay fatigue. Additionally, previous research has demonstrated that ingesting CHO that prevents a hypoglycaemic response prior to prolonged exercise may result in improved athletic performance by increasing blood glucose oxidation when intramuscular glycogen stores become compromised (Neufer *et al.*, 1987; Sherman *et al.*, 1991). Such meals that elicit these reduced blood glucose responses have typically been found to have a reduced glycaemic index.

The glycaemic index was first proposed by Jenkins *et al.* (1981) in order to describe the blood glucose responses to CHO foods. Previous research into the effects of manipulating the glycaemic index on exercise performance remains equivocal. Research has found consuming meals with a low glycaemic response ( $GI < 40$ ) to be associated with an improvement in exercise performance compared with the consumption of high GI meals ( $GI > 70$ ) (for e.g. DeMarco *et al.*, 1999; Kirwan *et al.*, 1998, 2001; Stevenson *et al.*, 2005; Thomas *et al.*, 1991). These observed improvements in exercise performance have typically been attributed to metabolic alterations that favour utilisation of fatty acids as a fuel, and a subsequent sparing of endogenous glucose and an increased hepatic glucose production late in exercise. However, the benefits of either a low or high

GI meal on exercise performance are inconsistent, based on studies showing no beneficial effect of these meals on TT performance or time to exhaustion during prolonged exercise (for e.g. Febbraio and Stewart, 1996; Sparks *et al.*, 1998; Thomas *et al.*, 1994; Wee, *et al.*, 1999).

A major limitation to the interpretation of previous research is that one or more aspects of the research design lacked some degree of internal or ecological validity. Some authors have attributed disagreements to small differences in study design, such as timing of the meal, exercise intensity, meal composition, and the specific metabolic responses to the meals, all of which may influence the metabolic and performance outcomes (Kirwan *et al.*, 2001). However, one of the most important aspects that limits the interpretation of previous research is the issue of defining and measuring 'performance'. Direct comparisons between studies cannot always be made, as some studies employed either a total work done in a fixed time protocol (Sparks *et al.*, 1998; Febbraio and Stewart, 1996), a time to exhaustion at a fixed work rate (Thomas *et al.*, 1991; Kirwan *et al.*, 1998, 2001; Stevenson *et al.*, 2005; Wu and Williams, 2006), which is associated with a high coefficient of variation with respect to time (McClellan *et al.*, 1995) and a lack of ecological validity, or employed a combination of the two methods (DeMarco *et al.*, 1999). Since performance is almost invariably measured as time to complete a fixed task the ecological validity of the performance measures used in previous studies is questionable.

The first two studies of this thesis therefore, aimed to address some limitations of the previous research by employing an exercise protocol that closely resembled that typically performed by competitive cyclists (TT) and providing a diet, consisting of real foods, which would typically be consumed prior to this type of performance. The findings offered support for previous research (DeMarco *et al.*, 1999; Kirwan *et al.*, 1998; 2001; Stevenson *et al.*, 2005; Thomas *et al.*, 1991; Wu and Williams, 2006) by observing that the ingestion of a low GI CHO meal 45min prior to performing a TT is associated with an improvement in endurance performance. Although the improvements in performance were similar to those previously observed, the metabolic responses observed in these two studies are not similar to those previously observed in similar studies. Instead of the typical inhibition of FFA and fat oxidation and increased rate of glycolysis associated with

the ingestion of high GI meals, CHO oxidation was actually greater for the low GI trial and fat oxidation was lower at this time. As endurance performance was improved, it was concluded, that the low GI meal may have contributed additional CHO for oxidation and subsequently spared limited muscle and liver glycogen stores as previously suggested (Sherman *et al.*, 1991; Kirwan *et al.*, 2001; Stevenson *et al.*, 2005). This additional CHO may have also provided a greater contribution of exogenous glucose to fuel use during the exercise bout as glucose was significantly higher at the end of the TT in the low GI trial compared to the high GI trial (Sherman *et al.*, 1991; Kirwan *et al.*, 2001; Stevenson *et al.*, 2005).

Additionally, the ability to perform prolonged, exhaustive exercise is closely related to muscle glycogen stores (Bergstrom *et al.*, 1967; Coyle *et al.*, 1986). Since athletes typically train or compete on consecutive days the rapid restoration of muscle glycogen stores during the recovery period is essential. Different types of CHO foods appear to have different effects on rates of muscle glycogen synthesis depending on the insulin and glucose response to the CHO load (Doyle *et al.*, 1993). The ingestion of high GI foods is generally associated with a rapid increase in blood glucose and insulin concentrations, whereas the ingestion of LGI foods results in a flatter, more sustained glycaemic response (Burke *et al.*, 1993). Since the rapidity of glycogen storage is influenced by both insulin and a rapid supply of glucose substrate, it is logical that CHO sources with a high GI would enhance post exercise glycogen resynthesis (Burke *et al.*, 2004). The rate of muscle glycogen storage in glycogen depleted muscle has been shown to be higher for the first 2h period when the participants ingested CHO immediately post exercise but lower when CHO consumption was delayed for 2h after the cessation of exercise (Ivy *et al.*, 1988). This increase rate is possibly because early post- exercise recovery is associated with an exercise- induced permeability of the muscle cell membrane to glucose and an increase in muscle insulin sensitivity (Burke *et al.*, 1998). Immediate CHO feeding takes advantage of these effects, slightly increasing glycogen storage rates and is most important when the interval between exercise sessions is short and the rate of refuelling must be maximized (Ivy *et al.*, 1988). Although the benefits of high CHO recovery diets are clearly

recognized, research into the possible influences of the different types of carbohydrates on subsequent performance is both limited and equivocal.

Additionally, although some research has been conducted into the effects of the ingestion of foods of different GI before exercise (Thomas *et al.*, 1991; 1994) and during 24h recovery periods following glycogen depleting exercise (Stevenson *et al.*, 2005), little research has been conducted into the effects of the GI of meals on shorter recovery periods between multiple bouts of exercise (Erith *et al.*, 2006). Previous studies investigating the effect of different types of CHO rich foods on glycogen storage have failed to find consistent results, possibly because research has taken a simplistic approach to CHO nutrition, dividing foods into “simple” or “complex” CHO foods on the basis of their chemical composition (Brewer *et al.*, 1988; Costill *et al.*, 1981; Roberts *et al.*, 1988). However, this system does not truly differentiate the effects of different CHO- rich foods on glucose and insulin responses. Also, many of the previous studies in this area have provided single foods in the hour before exercise, which is unlikely to reflect normal dietary behaviour and athletes are typically recommended to consume a high CHO meal 3-4h before exercise, and many recreational athletes will exercise at lunchtime 3-4 h after consuming breakfast (Hargreaves *et al.*, 2000). Therefore the last two studies of this thesis, aimed to investigate the influences that consuming high and low GI recovery diets has on both rates of recovery and subsequent performance, whilst trying to strengthen the methodologies of similar work in this area. For this, participants were provided with actual recovery diets, which were representative of foods and amounts that should normally be consumed by those types of athletes both prior to and following exhaustion exercise. Again the exercise test employed was similar to competition conditions, which these athletes would typically perform on a competition. The findings that emerged from the last two experiments in this thesis shared a similar conclusion. Both studies observed no difference in endurance performance following the consumption of either low or high GI recovery diets. This finding corroborates those of (Parkin *et al.*, 1997; Erith *et al.*, 2006), yet contradicts the findings of (Burke *et al.*, 1993; Stevenson *et al.*, 2005; Wee *et al.*, 2005). The outcome of these studies was that, provided that the availability and consumption of CHO is adequate for the athletes and is



provided within the 24h recovery window, the GI of the recovery meal appeared to have no further benefit/ improvement in endurance performance.

## **6.2 Practical Applications**

The aim of this research was to strengthen the methodologies of previous research in this area in the hope of applying the findings of a dietary intervention to more ‘real life’ sporting situations. Therefore, based on the outcome of the initial two studies of this thesis, it would appear reasonable to recommend the consumption of low GI meals, containing  $1\text{g.kg bw}^{-1}$  of CHO, 45min prolonged exercise would significantly improve their performance. The results from the two recovery studies of this thesis again aimed to strengthen past research within a similar area. The results of these two studies will promote the importance of adequate CHO consumption and availability within the 24h recovery period over the actual type of the CHO ingested. These studies demonstrate that, despite observed alterations in metabolism and oxidation rates, these differences did not confer into any performance benefit during either a long- term recovery (24h) or a more short- term recovery (3h) prior to performing a TT. These results support findings that 45min prior to endurance exercise, athletes should consume a low GI meal, providing  $1\text{g.kg.BM}^{-1}$  of CHO. However, following glycogen depleting exercise, athletes should focus more on consuming enough CHO during the recovery period to replenish depleted muscle glycogen stores, and less focus should be paid to the type of CHO they are ingesting during this time.

## **6.3 Limitations**

During most research, especially when working with athletes, it can be very difficult to control for all variables, therefore, there are limitations to the research within this thesis that must be considered when interpreting the findings.

The performance and metabolic outcomes of the participants used in these studies depend on participant compliance and whether or not the participants followed the same pre- testing

procedures and requirements before each trial. To control for this potential problem all participants were thoroughly briefed on the requirements of the study prior to participation and were also reminded of how important it is that they conform to the requirements on the pre- exercise checklist and follow them strictly. The participants were also required to sign that they had followed the requirements of the checklist and required baseline data to ensure that they had not eaten and they were fully hydrated prior to exercise. Any participants that did not follow these criteria were asked to leave the laboratory and return at another date, when they had followed the required criteria.

Although this research was trying to represent as closely as possible, the athletic conditions associated with cycling TT performance, there are several aspects of the research, which, unfortunately decrease the ecological validity. These include collecting both gas and venous blood samples from the participants at various time points throughout the TT, which would obviously not typically be done during a TT competition. Additionally, the participants were only allowed to drink water both prior to and during the TT. Again, most athletes would be typically consuming sports drinks or CHO containing fluids prior to and during endurance exercise. However, recent research (Backx *et al.*, 2007) has observed that despite an availability of fluid, cyclists chose to drink during a 40 k TT race with no negative effects on race time being apparent, suggesting that this may not be a major limitation to the study.

The athletes' diets were monitored for two days before each trial to establish that the energy and macronutrient intake were similar. However, two days might not have been of sufficient duration to negate the confounding residual effects of the pre-experimental intervention diet on the physiological and performance responses to the exercise. In addition to this, the 24h recovery diet was provided to the participants so it was therefore, up to them to consume the meal whilst they were at home and not to deviate from their prescribed 24h recover meal. However, although again the participants were required to sign that they had followed the diet, again, there is no way of knowing that this was strictly followed.

Finally, the glycaemic and insulinaemic responses to the recovery diets were not measured during each recovery period. Knowing this information may have demonstrated the metabolic

effects that each meal was initially having on substrate availability and recovery and provided more information about the initial recovery immediately after the cessation of exercise. In addition, the ability to perform muscle biopsies would have also provided us with much more interesting information into muscle glycogen synthesis recovery rates and which meals were associated with the greatest rates of muscle glycogen recovery, rather than speculating from CHO and fat oxidation data. However, as this was not measured it was not possible to identify which meal was associated with the greatest initial rates of glycogen recovery.

#### **6.4 Recommendations for Future Research**

The athletes involved in this research did not consume a CHO drink during the exercise trials so that the effect of the GI content of food was not confounded by the physiological responses to the CHO drink. However, since athletes typically ingest a CHO drink during competition, the moderating effects of a CHO drink on the effects of the GI content of food on athletic performance has important practical implications. This problem should and could be addressed in future research by using a three-way experimental design, using 'drink' as an additional factor.

Future research in this area should also consider the number of days the diet is controlled leading up to the trial and might consider a longer monitoring or dietary control period. In addition to this, in order to control the athlete's intake prior to and following exercise, it may be better if there was a place for the participants to reside during the duration of the study, so that all the variables can be controlled for.

Future research also needs to focus on strengthening the methodologies of previous research and to focus on more popular/ real sporting situations. Also, more invasive methods should be employed in the protocol such as the use of muscle biopsies to determine, more effectively, which diet, if any is having more influence on muscle glycogen resynthesis following exhaustive exercise.

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## Appendices

**Appendix 1: An example of a Pre- exercise Medical Form**

The information in this document will be treated as strictly confidential

Name:

.....

Date of Birth: ..... Age: .....Sex: .....

Blood pressure: ..... Resting Heart Rate: .....

Height (cm): ..... Weight (Kg): .....

**Please answer the following questions by putting a circle round the appropriate response or filling in the blank.**

1.

How would you describe your present level of **exercise** activity?  
Sedentary / Moderately active / Active / Highly active
2.

Please outline a typical weeks exercise activity  
  
.....  
.....
3.

How would you describe your present level of **lifestyle** activity?  
Sedentary / Moderately active / Active / Highly active
4.

What is your occupation? .....
5.

How would you describe your present level of fitness?  
Unfit / Moderately fit / Trained / Highly trained
6.

Smoking HabitsAre you currently a smoker?Yes / No  
How many do you smoke..... per day  
Are you a previous smoker?Yes / No  
How long is it since you stopped?..... years  
How many did you smoke?..... per day
7.

Do you drink alcohol?Yes / No  
  
If you answered **Yes** and you are male do you drink more than 28 units a week?  
Yes / No  
  
If you answered **Yes** and you are female do you drink more than 21 units a week?  
Yes / No
8.

Have you had to consult your doctor within the last six months?Yes / No  
If you answered **Yes**, Have you been advised **not** to exercise?  
Yes / No
9.

Are you presently taking any form of medication?Yes / No  
If you answered **Yes**, Have you been advised **not** to exercise?  
Yes / No
10.

To the best of your knowledge do you, or have you ever, suffered from:  
  
a Diabetes?Yes / No      b Asthma?Yes / No

|   |          |                                 |          |
|---|----------|---------------------------------|----------|
| <b>c</b> Epilepsy?                      | Yes / No | <b>d</b> Bronchitis?            | Yes / No |
| <b>e</b> ★ Any form of heart complaint? | Yes / No | <b>f</b> Raynaud's Disease      | Yes / No |
| <b>g</b> ★ Marfan's Syndrome?           | Yes / No | <b>h</b> ★ Aneurysm / embolism? | Yes / No |
| <b>i</b> Anaemia                        | Yes / No |                                 |          |

11. ★ Are you over 45, and with a history of heart disease in your family? Yes / No
12. Do you currently have any form of muscle or joint injury? Yes / No  
If you answered **Yes**, please give details.....  
.....
13. Have you had to suspend your normal training in the last two weeks? Yes / No  
If the answer is **Yes** please give details.....  
.....
14. ★ Please read the following questions:
- |    |   |          |
|----|---|----------|
| a) | Are you suffering from any known serious infection?   | Yes / No |
| b) | Have you had jaundice within the previous year?   | Yes / No |
| c) | Have you ever had any form of hepatitis?  | Yes / No |
| d) | Are you HIV antibody positive   | Yes / No |
| e) | Have you had unprotected sexual intercourse with any person from an HIV high-risk population? | Yes / No |
| f) | Have you ever been involved in intravenous drug use?  | Yes / No |
| g) | Are you haemophilic?  | Yes / No |
15. As far as you are aware, is there anything that might prevent you from successfully completing the tests that have been outlined to you? Yes / No.

**IF THE ANSWER TO ANY OF THE ABOVE IS YES:**

- a) Discuss with the test administrators or another appropriate member of the department.**
- b) Questions indicated by ( ★ ) answered yes: Please obtain written approval from your doctor before taking part in the test.**

**PLEASE SIGN AND DATE AS INDICATED ON THE NEXT PAGE**

Participant Signature: .....Date.....

Test Administrator:.....Date.....

Parent if Minor.....Date: .....

## Appendix 2: An example of an Informed Consent Form

### Informed Consent Form

Name of test(s).....

#### **The participant should complete this sheet himself / herself**

- |   |          |
|---|----------|
| 1. Have you completed the pre-exercise medical questionnaire?               | YES / NO |
| 2. Do you understand that your information will be treated as confidential? | YES / NO |
| 3. Have you read the participant information sheet?                         | YES / NO |
| 4. Have you had the opportunity to ask questions and discuss the test?      | YES / NO |
| 5. Have you received satisfactory answers to all of your questions?         | YES / NO |
| 6. Have you received adequate information about the test?                   | YES / NO |
| 7. With whom have you discussed the nature of the test?                     |          |

.....

- |   |          |
|---|----------|
| 8. Do you understand that you may withdraw from the test: |          |
| • At any time   |          |
| • Without needing to give reason                          |          |
| • Without prejudice                                       | YES / NO |

9. I have read, discussed and fully understand the requirements, procedures, and potential risks involved in the test and give consent for my participation.

Signature..... Date.....

Test Administrator..... Date.....

Parent/Guardian if Minor..... Date.....

### Appendix 3: General Pre-Test Procedures and Check List Form

#### Confirmation checklist - General pre-test procedures

Name ... Date ..... Test .....  
Contact details... Mob..... Home.....

**Please tick the relevant box for each statement.**

1. I **am** wearing the same clothing and training shoes that I was wearing for my last test (tick if applicable).  
True ☐ False ☐
2. I **have** drunk plenty of fluids during the last 24 hours, including at least 500ml of water approximately 2 hours ago.  
True ☐ False ☐
3. I **have not** eaten since for >6 hours  
True ☐ False ☐
4. I **have not** consumed alcohol or marijuana in the last 24 hours.  
True ☐ False ☐
5. I **have not** consumed caffeine, nicotine or ephedrine in the last 8 hours.  
True ☐ False ☐
6. I **am not** suffering from any **injury/muscle soreness/infectious illness**.  
True ☐ False ☐
7. I feel alert and mentally and physically ready for the test I am about to take part in.  
True ☐ False ☐
8. I will return for my re- trial on the (date).....

Signature .....

**Thank you for taking the time to complete this form.**